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ON THE VARIABILITY OF MIXED RACES

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THE PROBLEM

IN 1927 Herskovits called attention to the important fact that the American Negro population studied by him did not have an appreciably greater variability in respect to most characters than did populations of whites and African Negroes related to those which had gone to form the mixed group. The mixed group in question was probably derived to the extent of at least 20 per cent. (possibly much more) from Caucasian stocks, and also had a small indeterminable amount of American Indian admixture. The mixed group was roughly divided by him into several subgroups, according to the relative proportions in which the two races had entered into the individual's composition (as determined by questions concerning the individual's ancestry) and in each of the subgroups (even in that consisting of more nearly 50 per cent. from each of the two races chiefly concerned) the low variability was found. Herskovits concluded that "the Mendelian mechanism of heredity, if operative at all in this population, is obscured by complicating factors." He was inclined to think that through the process of repeated breeding of the members of the mixed race amongst one another, a true new race of intermediate type had become established, the stability of which was quantitatively similar, and (presumably) similar in its basis to the stability of the original races. In a more

recent article (1934) he repeats that "obviously it takes many generations to produce such community of type in the mixed population."

In this more recent article, Herskovits is at pains to point out that Mendelian heredity may be operative here, especially Mendelian heredity "of a complex type," but says that "it is when it comes to scientific proof that faith in the truth of such a statement must remain in the realm of faith." It is evident, however, to the student of Mendelism that Mendelian heredity, no matter how complex, should not result in lesser variability after twenty generations of interbreeding of the mixed population amongst one another than after only two (supposing that the first generation had consisted exclusively of a series of crosses between the two original races*), and Herskovits himself has no Mendelian explanation to offer of the phenomenon in question.

The unincreased hybrid variability brought to the fore by Herskovits's work has also been illustrated in the work of other investigators, both before and after him (see review by Wagner, 1932). Thus, Eugen Fischer in 1913 had made similar findings in his study of the "Rehobothe Bastarde"—a population of mixed Hottentot and Dutch ancestry. But Fischer, a strong Mendelian, had laid little stress on this result and was inclined to blame it upon some unsuitability of the mathematical measure of dispersion used (the standard deviation coefficient, σ). This stricture, however, can hardly apply, since the term variation hardly has any exact scientific meaning except when defined as the standard

* In the case of a population containing a non-random distribution of genes at different loci with regard to one another (such as would be produced in the parental generation by mixing together the individuals of two different races) a state of random distribution of these genes in relation to one another is only gradually approached in the course of successive generations of purely random interbreeding, as shown by Jennings, Wentworth and Remick, Robbins, Philipchenko, etc. But if we consider as F_1 only the products of crossing between the races, we find that the random distribution is (except in case of linkage) already attained in F_2 from the intercrosses of F_1, and it is of course maintained thereafter.

deviation itself, or as its square (the variance), or as the standard deviation divided by the mean (the coefficient of variability). And on any customary Mendelian assumptions, such as have been made hitherto in considerations of race crossing, the standard deviation would be greater in the F_2 and later generations from a crossing between two different (relatively homozygous) stocks than within these stocks themselves. As above pointed out, however, it would be no different in F_2 than in the later generations.*

Davenport also had obtained some similar results in his study of the mixture of whites and Negroes in Jamaica, but he believed that the lack of increased variability applied only in the case of characters in regard to which the original races were phenotypically very similar. As Wagner has shown, however, the results seem to be not very different in regard to most measured characters that distinguish the races. Moreover, a phenotypic resemblance between the races in regard to a certain character is no sound indication of their genotypic resemblance in regard to this character (*cf.*, for instance, Harland's work on cotton), and hence, on the ordinary Mendelian view, even those characters which were phenotypically alike in the two races should often show considerable segregation, that is, increased variability in the mixed race. Davenport further suggests (in the case of the Rehobothe Bastarde) that the variability of the mixed race may have become appreciably reduced, within the four or five generations of the interbreeding, by the operation of natural and sexual selection. This seems to us, however, extremely improbable, especially when it is remembered that even pure whites perpetuate themselves well in this region, and that the hybrid people in question still show mean values that are distinctly intermediate.

In Shapiro's recent work on a mixed population on Norfolk Island, derived from English and Tahitians, in which a similar low variability was found, the investigator interprets the result as a consequence of the preva-

lence of inbreeding, cousin marriage being very common. Here again, the explanation is contrary to Mendelian principles, for inbreeding should increase rather than decrease variability in the population as a whole, inasmuch as the latter would then tend to be formed of a series of different inbred lines, more or less independently derived from the cross between the two races.

It must be admitted that in most of the works which have touched on the general problem in question, the data reported are hardly satisfactory for an accurate comparison of the variability of the mixed race with the original races, in regard to characters in which the latter differed strongly from one another. For one thing, when the standard deviations are given, the mean values are often omitted, so that it is not evident whether the difference between the means is large compared with the standard deviations concerned, and whether there is much phenotypic overlapping of the original races. Moreover, in some of the data, individuals resulting from a primary cross between the two races, that is, F_1's, are not always rigorously excluded; their inclusion should unduly lower the variability of the mixed group. On the other hand, the fact that the individual members of the mixed group have in some of the cases different amounts of one or the other original race in their ancestry tends to increase the variability of the mixed group; this has been only partly taken care of, in most cases, by roughly dividing the mixed race into three subgroups having different proportions of the two original races in their composition. Again, the original races themselves contain various subgroups, whose means and standard deviations often differ considerably from one another, and data on that exact subgroup, or combination of subgroups, which really entered into the composition of the mixed race, have seldom been available. The fact that the mixed race is usually a mixture not merely of the two main races but also of various subgroups of the latter, might be expected to increase its variability, in comparison with

that of any one subgroup of one of the original races: this stricture is especially applicable in cases in which the products of these different mixtures may not have undergone quite random crossing among one another, in the later interbreeding of the mixed race.

But even taking into account these deficiencies of the data, it must be admitted that, were there a marked tendency for increased variability in mixed races, this tendency should have shown more clearly in the investigations already reported. In several cases in particular, data were given showing a strong difference between the means of the two original main races, and relatively little difference between the means of the subgroups of either race, and in all these cases, although F_1's formed no appreciable part of the mixed race, the variability of the mixed race or subgroup of the latter was certainly not much higher than that of either original race. While, therefore, further data are desirable, in order better to define the results in quantitative terms, the problem is a real one and demands interpretation, and this interpretation must of course be Mendelian, since it is unthinkable that the processes of heredity in man are not Mendelian, just as they have been proved to be in all other animals that have been investigated with genetic exactitude.

Provisional Interpretation

There are several circumstances which could give rise to a comparatively low variability on the part of a population of mixed race, as compared with the original populations which went to form the mixture, without the slightest violation of Mendelian or other accepted genetic principles being involved. One of the possible mechanisms by which such an effect could be produced would involve the partial suppression, in the mixed race, of the intraracial variability of either original race. This suppression might conceivably be so great as to fully compensate, or more than compensate, for that part of the

variability of the mixed race which was caused by the segregation from one another of those genes that had caused the two original races to differ. Thus the intraracial variability of the new mixed race would be largely constituted of the *inter*racial differences, and the original *intra*racial differences would now be in part suppressed in their expression.

It may be best at first to illustrate the principle here involved by a simple hypothetical example. Suppose that in one of the original races, race "1," the character under consideration, let us take, for example, the breadth of the nose, was subject to variation owing to the presence in some individuals of a certain recessive gene, *a*, that tended to increase the width of the nose relatively to that of individuals having the "normal" gene *A*. Let us assume that in regard to the locus in question, 75 per cent. of the genes in race 1 had the "normal" allelomorph *A* and the other 25 per cent. the recessive "abnormal" (probably mutant) allelomorph, *a*; p_1, the proportion of recessive genes at this locus in race 1 thus becomes ·25. In that case, if the matings within race 1 were at random, the proportion of homozygous *a individuals* would of course be p_1^2 or .0625 (1/16), since the chance of an individual having received *a* from both egg and sperm is the product of the chance, ·25, that *a* should have been in the egg, by the same chance, ·25, that it should have been in the sperm. Now let us suppose that in the other original race, "2," the recessive gene *a* practically does not exist; that is, in race 2, *p* has zero frequency ($p_2 = 0$). This second race, however, will probably have about as variable a nose as the first race, even though the mutations that cause its variation lie in other loci. To simplify again, let us suppose that in race 2 there is another recessive gene, *b*, similar in its effect to the *a* of race 1, and that its frequency likewise is ·25, in race 2. Thus the frequency of homozygous *b* individuals in race 2 (like the frequency of homozygous *a* individuals in race 1) will be ·0625 or 1/16. Race 1, on the other hand, we may

suppose to be virtually free of the gene *b*, containing only the normal allelomorph *B*. Expressing this more fully, then, we may say that $p_{1a} = .25$; $p_{2a} = 0$; $p_{1b} = 0$; $p_{2b} = .25$.

Now when these two races, both having the same amount of variability, are crossed together to form a mixed race, what will be the result? If the mixed race is made up of equal amounts of genes from the two original races, assorted at random as they would be in F_2 and in all later generations of a random-breeding mixture, the concentration of gene *a* in the mixed race must of course be half of what it was in race 1 (since race 1 is the only source from which it came), and the concentration of gene *b* must similarly be only half of that in race 2. That is, using the letter M to represent the mixed race, p_{Ma} is ⅛ or ·125 and p_{Mb} is also ⅛ or ·125. Individuals homozygous for *a* in the mixed race, that is, those exhibiting the abnormally wide nose caused by *aa*, will now be present in the proportion $(p_{Ma})^2$ or $(⅛)^2$ or 1/64 or .015625. The same is true of the homozygous *b* individuals. The two classes together, homozygous *a* and *b*, which are indistinguishable from one another, will constitute two times 1/64; that is, 1/32, or ·03125. Of course there will be a little genetic overlapping here, that is, some of the homozygous *a*'s will at the same time be homozygous *b*'s, there being in fact 1/64 × 1/64 of the combinational class and each of the singly homozygous classes being reduced by this amount; however, if the effects are cumulative the variance of the population will be little affected by this small amount of overlapping. Thus we see that, whereas in either original race 1/16 of the individuals showed an abnormally wide nose, in the mixed race a little less than 1/32 of the individuals, that is very nearly half as many as in either original race, show this peculiarity. Similar relations are shown by the variance of the mixed race, in comparison with the variance of the original races, *i.e.*, the former is very nearly (but a little more than) half of the latter, while σ (the square root of the

variance) in the mixed race is about ·7 that in the original races.

While the above of course represents a simplification, nevertheless the same principle would operate in the case of any other recessive genes that were present in not too high a frequency in one original race, and in negligible frequency in the other original race, and in so far as the total intraracial variability of either of these races had been caused by such genes, acting in a cumulative way, the variance of the population of mixed race would be approximately halved. At the same time, however, the two original races might differ from one another in the mean values of their nose-width by reason of certain loci in regard to which race 1 was homozygous (or nearly homozygous) for one allelomorph, while race 2 was nearly homozygous for the alternative allelomorph. These genes would give rise to little or no variation in either original race, being virtually homozygous in them, but in the mixed race their segregation would give rise to variation in F_2 and in all later generations, and this cause of increased variation would tend to compensate for the above-mentioned cause of decreased variation, so that the mixed population might be left with about the same resultant variability as either original race had had. This should make clearer the meaning of our previous statement, that in the mixed race the intraracial variability was derived in part from the segregation of interracial differences, while at the same time the *intra*racial variability of the original races had been in part suppressed in the mixed race, the two tendencies more or less compensating one another.

It now remains to generalize upon this interpretation. Expressions should be found stating in quantitative terms the variability of the mixed race in comparison with that of the pure races, under various possible conditions of dominance and of relative gene frequency in the two races. We shall then be in a better position to know how special the conditions must be which would

give rise to a relatively low variability of the mixed race. And in the course of this formulation, we shall see that other possible interpretations of the low variability will also emerge.

The Relation of Variance to Gene Frequency and Dominance

The problem at issue requires, first, that we should have an expression whereby we can calculate the variance, standard deviation or coefficient of variability of any population, on the assumption of any given gene frequency and degree of dominance. The calculation may be made for the case of a single locus, and this can readily be used for determining the effects of given combinations of loci on the provisional assumption that the combined effects of the genes in different loci are additive, for in that case the variance due to the different loci will simply be the sum of the variances due to each of the loci taken separately.

Confining ourselves to the given locus, then, and considering only a single pair of allelomorphs at that locus (*i.e.*, excluding for the sake of simplicity the possibility of multiple allelomorphism), let us as before represent by p the proportion of genes of the recessive (or of the more nearly recessive) type present in the population. $1-p$, which may alternatively be represented by q, will then be the proportion of the dominant (or of the more nearly dominant) genes of that locus. In random breeding, as Hardy first pointed out, the number of individuals respectively homozygous recessive, heterozygous and homozygous dominant will be represented by the formula $p^2 + 2pq + q^2$. From this the mean and the variance of the population may readily be found, if we can give numerical or algebraic expressions for the phenotypic values of the three classes mentioned. The most useful mode of expression for this purpose is to give a value of zero to one of the homozygous classes, let us say to the class of homozygous dominants, and a value of 1 to

the other homozygous class (the recessives). The heterozygous class may then be given a value of h, the latter being a fraction ranging from zero to ·5 according to the degree of dominance, zero representing the condition of complete dominance and recessiveness, and ·5 the condition of exact intermediacy of the heterozygote, *i.e.*, no dominance.

To find the mean, then, we have only to multiply the frequencies of the individuals in each class by their respective phenotypic values (division of the product by the total number of individuals in the population being here unnecessary, as the frequencies are here represented as proportions rather than whole numbers, so that the population number becomes 1). Making these multiplications, we have for the mean:

$$p^2(1) + 2pqh + q^2(0)$$

which reduces to:

$$p^2 + 2pqh \qquad \text{(Formula 1)}$$

The variance is of course the sum of the squares of the deviations of the phenotypic values of each individual in the population from the mean value, divided by the number of individuals in the population, and according to a familiar formula this may be obtained by first finding the sum of the squares of the deviations of the phenotypic values of each individual from the zero value, dividing this sum by the number of individuals in the population (in this case 1), and then subtracting from this value the square of the mean value. Applying this to our problem, each of the class frequencies given by Hardy's formula must first be multiplied by the square of the phenotypic value which we have assigned to that class. These products are then added together, and (division by 1 being superfluous) from this sum there must be subtracted the square of the value for the mean given in Formula 1. This gives us:

$$p^2(1^2) + 2pqh^2 + q^2(0^2) - (p^2 + 2pqh)^2$$

which reduces to:

$$\sigma^2 \text{ (the variance)} = p^2 + 2pqh^2 - (p^2 + 2pqh)^2 \qquad \text{(Formula 2)}$$

The standard deviation itself, σ, is merely the square root of this.

This formula was then applied to the construction of tables showing the means and variances of populations having any value of p from zero to 1, progressing by intervals of ·05—*i.e.*, for the values, ·05, ·10, ·15, ·20, etc. This was done in the case of the following values of h: 0, ·1, ·2, ·3, ·4 and ·5, a separate column being devoted to each of these six values of h, showing the values of M and of σ^2 corresponding with each of the values of p indicated on the left. The results for σ^2 are shown in Table A. (The values of M are the same as the lowermost figures in the squares forming the extreme right and left columns of Tables B to G, inclusive.)

TABLE A

VARIANCE (σ^2) CAUSED BY PRESENCE IN POPULATION OF RELATIVELY RECESSIVE GENE HAVING DIFFERENT FREQUENCIES (p) AND DEGREES OF DOMINANCE (h). PHENOTYPIC VALUE OF HOMOZYGOUS RECESSIVE IS TAKEN AS 1, OF HETEROZYGOTE AS h, AND OF HOMOZYGOUS DOMINANT AS 0

p	h = 0	h = .1	h = .2	h = .3	h = .4	h = .5
0.00	.000000	.000000	.000000	.000000	.000000	.000000
.05	.002494	.003306	.005838	.0100890	.016060	.023750
.10	.009900	.011016	.015084	.0221040	.032076	.045000
.15	.021994	.022746	.027298	.0356490	.047800	.063750
.20	.038400	.038016	.041984	.0503040	.062976	.080000
.25	.058594	.056250	.058594	.0656250	.077344	.093750
.30	.081900	.076776	.076524	.0811440	.090636	.105000
.35	.107494	.098826	.095118	.0963710	.102580	.113750
.40	.134400	.121536	.113664	.1107840	.112896	.120000
.45	.161494	.143946	.131398	.1238490	.121300	.123750
.50	.187500	.165000	.147500	.1350000	.127500	.125000
.55	.210994	.183546	.161098	.1436490	.131200	.123750
.60	.230400	.198336	.171264	.1491840	.132096	.120000
.65	.243994	.208026	.177018	.1509690	.129880	.113750
.70	.249900	.211176	.177324	.1483440	.124236	.105000
.75	.246094	.206250	.171094	.1406250	.114844	.093750
.80	.230400	.191616	.157184	.1271040	.101376	.080000
.85	.200494	.165546	.134398	.1070490	.083500	.063750
.90	.153900	.126216	.101484	.0797040	.060876	.045000
.95	.087994	.071706	.057138	.0442890	.033160	.023750
1.00	.000000	.000000	.000000	.0000000	.000000	.000000

For the purpose of our present problem, we should now find the variance or standard deviation of a mixed

race for comparison with those of the original races that went into the mixture, on the assumption of various values of p (gene frequencies) in the two original races and of various values of h (dominance). We may assume the most extreme possible mixture, that is, one of equal parts of the two original races, the result in any other case being intermediate between the result for the more heavily represented original race alone and for the equally mixed race.

Table A was therefore used for the construction of further tables, showing the values of the variance (σ^2) of a mixed race as compared with the average variance of the original races, in the case of the different possible values of p and h. p_1, the value of p in race 1, was allowed to vary from 0 to 1.0 by steps of 0.1, and p_2, the value of p in race 2, was allowed to vary independently in the same way. p_M, the value of p in the equally mixed race, was of course halfway between p_1 and p_2, *i.e.*, $p_M = \frac{p_1 + p_2}{2}$.

Since now our problem was to find out how, under each set of gene frequencies, the variability of the mixed race was increased or decreased relatively to the original races, it was necessary for us, after looking up in our table A the variance which the mixed race would have, to subtract from this the value representing the average variance of the original races. This difference was then divided by the latter value itself (that is, by the average variance of the original races). We may call the resultant ratio r: *the relative increase of variance* of the hybrid population, caused by the gene pair in question. At the same time it is important to note how much variance the original races had on the average manifested in the given case, as a result of their heterogeneity in regard to this locus, and the extent to which the mean of one original race had differed from the mean of the other as a result of the same gene pair.

The results in question are shown in our tables B to G. In these tables the numbers in the extreme right or left-hand column show the assumed values of p ("p_2") in one of the original races, and the numbers in the row along the top or bottom show the corresponding values of p ("p_1") in the other original race. In each rectangle formed by the intersection of the vertical column leading from the first value of p with the horizontal row leading from the second value of p, we find r, the relative increase in variance of the mixed race resulting from the crossing of the races having these two respective values of p. This value is the uppermost figure shown in each rectangle; it is reckoned to an accuracy of $\pm$·001. Below it, in the middle of the rectangle, is shown the average variance of the assumed original races, caused by this locus. The lowermost figure in each rectangle represents the difference between the means of the original races, in so far as the locus in question affects these means. Only about half of each table is presented, the part omitted being in each case a mirror-image of most of the portion shown, and having the given values of p_1 and p_2 interchanged for one another; *i.e.*, in the full table the same values would occur on both sides of the diagonal line of rectangles leading from the upper left to the lower right hand corner, and they would occur symmetrically distributed with regard to that line. This diagonal bisecting line may be called "the line of zero differences"; the regions farthest from this line (the upper right and the lower left corners) are the regions of maximal differences.

Before going on to consider the figures in these tables, a few words may be said concerning our use of variance (σ^2) here instead of standard deviation (σ). We have chosen variance as the more convenient to use because of the fact that the variance due to different loci may be added together for the finding of the total resultant variance, when the effects of the genes at the different loci are additive. The same can not be done with standard

deviation. Unfortunately, the average standard deviation of the two original races bears no constant relation to their average variance and the one can not be calculated from the other. But there is an ameliorating consideration by virtue of which our tables of variance come to have more meaning in relation to standard deviation. This is the consideration that, on the whole, the same general principles of mutation and of differential multiplication have probably applied to both original races, so that in most cases both would have come to have a similar amount of variability, based upon heterozygosity in similarly acting genes which, though they may have been at different loci, nevertheless tended to have a similar distribution of frequencies. Thus, if the variance be due to very many genes and if we suppose a certain part of the variance in the two races to be due to genes which in one race have a certain frequency and in the other race another frequency ($p_1 = a$, $p_2 = b$), then we may suppose it to be likely that there will probably be other genes, about as important as the former, which in the first race have approximately the latter frequency and in the second race approximately the former frequency ($p_1 = b$, $p_2 = a$). Then the resultant variance, due to both groups of genes, will be about the same in the two original races, and so the "average" variance of the two races will also have this same resultant value; likewise the two original resultant standard deviations and the average resultant standard deviation of the two original races will be about equal to one another. Now in this case "σ_2," the average resultant standard deviation of the original races due to both sets of genes (both to those having the frequency $p_1 = a$, $p_2 = b$, and to those of frequency $p_1 = b$, $p_2 = a$), will bear a fixed relation to "σ_2^2," the average resultant variance due to both sets of genes, and also to the simpler value σ^2 shown in our tables, *i.e.*, to the average variance due to one set of genes. The relation of σ_2 to σ^2 is simply:

$$\sigma_2 = \sqrt{2\sigma^2} \qquad \text{(Formula 3)}$$

as may easily be shown.

Furthermore, the ratio r, if calculated on the basis of the standard deviation resulting from both sets of genes and then designated as "r_{σ_2}" will bear a fixed relation to the corresponding "$r_{\sigma_2^2}$" calculated on the basis of the variance resulting from both sets of genes, and also to the simpler value which has above been denoted as r and used in our tables, and which is calculated on the basis of σ^2, the variance caused by one set of genes. The relation of r_{σ_2} to our r is as follows:

$$r_{\sigma_2} = \sqrt{2r+1} - 1 \qquad \text{(Formula 4)}$$

These formulae can be used, where desired, for the conversion of our tables into a form involving standard deviation, in cases in which it is feasible to accept the above assumption regarding the symmetrical distribution of gene frequencies in the two races. For cases in which it is not feasible to accept this assumption, we may note that tables of σ and of r_σ, *i.e.*, of the average standard deviation of the original races and of the value of r based on standard deviation, when only *one* set of genes (one locus) is taken into consideration, have been published in our Russian presentation of this subject.[1] The latter tables, then, correspond with those here published, except that they are based upon standard deviation instead of upon variance, and there are no formulae possible for the conversion of the values in those tables into those of our present tables.

Table B represents the values under the condition of complete dominance ($h = 0$). An examination of this table shows that in about one third of the different possible combinations there is a negative value of r, that is, the variability of the hybrid population is actually lower than the average variability of the original populations, in so far as these variabilities are due to the genes in question. These negative values are greatest where the frequency of the recessive gene is rather low in both races, and where it is at the same time considerably lower

[1] *Bull. Medicogen. Inst.*, 4: 1936.

in one of the races than in the other. Our simple example considered in the previous section falls into this category. This is the group of cases *par excellence* in which an actual decrease of hybrid variability occurs. As the frequency of the recessive gene in one race becomes smaller and as the frequency of the recessive gene in the other race at the same time diminishes relatively to its frequency in the first race, the value of r approaches $-.5$; that is, in the limiting case, the variance is halved in the hybrid. The results are seen to approximate these limiting negative values in all cases in which p_1 is less than .5 and p_2 is 0. Referring now to the figures for average variance in the middle of the rectangles, we see that there is not a great deal of variability produced in the original races when the frequencies of p are very low, and hence the absolute decrease of variability in the hybrid, caused by one locus in which these frequencies occurred, would not be great, even though r has a relatively high negative value in these cases. A large absolute decrease of variability could however be produced by means of the combined action of many loci, all of which met these conditions, that is, by many genes which had a low frequency in one of the original races and a very much lower frequency still in the other original race. But it is exactly such genes which in the current theory of heterosis are postulated to be numerous, and considerations arising from modern work concerning mutations also lead to the conclusion that they are numerous, relatively to genes having other frequencies.

When p_1 becomes so great that approximately half of the individuals in race 1 manifest the recessive character, r sinks to zero. More accurately, r becomes exactly zero when p_1 rises to .757, in case p_2 is zero (*i.e.*, when the value of p_2 is most favorable for negative values of r). For higher values of p_1 than this, r becomes positive, that is, there is an increase in the hybrid variability above that of the average of the original races.

Near the diagonal "line of zero differences" is a group of rectangles representing cases in which the frequencies of the genes in question are similar in the two original populations crossed (*i.e.*, in which p_1 is nearly equal to p_2). It will be seen that this diagonally situated group of rectangles has values of r which approximate zero throughout (the values of course become exactly zero when p in the two races becomes exactly the same; therefore the median diagonal line of zero differences also has exactly zero values for r). The values of r in those rectangles near the upper left-hand part of the line are on the negative side of zero and in those near the lower right-hand part are on the positive side, but the deviation from zero is in many cases hardly great enough to cause differences which could readily be established by such statistical studies of populations as have been made. Here then is a group of cases in which, although the variability of the hybrids is not actually decreased, nevertheless it is not increased markedly either. Hence, if most of the variation seen in the original races, *as well as* most of the differences *between* these races, were caused by genes of this type (genes having very similar frequencies in the two races), the lack of increased variability in the hybrid would be sufficiently explained without recourse to genes having such frequencies as actually to decrease the variability strongly. It seems, however, rather arbitrary to assume that most of the differences between two major races should be caused in such a manner; reasons for this will be adduced later.

In the upper right-hand corner of the table and in the rectangles nearby, especially in those lying below it and not too near to the diagonal line above discussed, we find very high positive values of r, that is, large increases of hybrid variability. These represent cases in which the two original populations may be considered as relatively homozygous for the opposite allelomorphs of the locus concerned. If (as is more usually assumed) much of the difference between the means of the original races

were caused by such genes, then there would be a considerably increased hybrid variability, due to this cause, *unless* this increase were compensated for by the simultaneous existence of a large class of cases giving relatively high negative values of r, that is, by the cases of low-frequency recessive genes first discussed, which fall in the upper left-hand portion of this table.

Passing over now to the table representing the opposite extreme, the condition of no dominance at all, table G, we notice that there are here no negative values of r, that is, no conditions under which the hybrid variability could show any decrease. As before, however, the values near the diagonal line, representing a condition of similarity in the frequencies of p_1 and p_2, approach zero. Contrariwise, the values become greater as we draw nearer the upper right-hand (or its mirror-image, the lower left-hand) corner, the region of the table representing very different values of p_1 and p_2, *i.e.*, conditions in which one race is relatively homozygous for one allelomorph and the other race for the other allelomorph. If all genes were non-dominant, then, there could be no actually decreased variability, but there might be a not greatly increased variability in the hybrid, provided the frequencies of the genes were, in the case of most loci having an important influence, very similar to one another in the two races. But it is to be noted that this condition would have to apply even in the case of those genes which chiefly were instrumental in causing the means of the two original races to differ from one another, if these genes exerted a large influence. If some of the important loci that served to differentiate the two original races differed considerably in their gene frequencies in the two races (as would be the case to the most extreme degree when the two races were homozygous for opposite allelomorphs), this difference should be manifested in the hybrid by an appreciably increased variability, since in the case of non-dominance there is no

possibility of other loci producing a decreased variability to compensate for the increase caused by the former.

Perusing now the tables dealing with intermediate grades of dominance, between zero and .5, we find of course that the results are intermediate between those of Tables B and G. But only in the case of $h = .1$ or .2 is any really appreciable degree of *decrease* in variability to be found. In the case of $h = .3$, r never attains a negative value greater than about $-.12$; this could hardly exert any considerable effect in compensation for an increased variability caused by the original races being relatively homozygous for opposite allelomorphs at other loci. When $h = .2$, the decrease of variability is appreciable, but here the degree of dominance (and recessiveness) may already be considered fairly high, inasmuch as one gene here has four times the potency of the other gene, in determining the phenotype of the heterozygote (the latter standing at .2 units from one homozygote and at .8 from the other). Thus we see that it is only under conditions (1) of fairly "good" dominance (and recessiveness) that an actual decrease in variability can be produced, and then only when (2) the recessive genes causing the variation in one race have rather low frequencies, and (3) when the same genes in the other race have frequencies relatively much lower still.

Discussion

On that explanation of the lack of increased variability in hybrid races which seems for the present the more plausible, we reach the following provisional conclusions concerning the variability that is present within the original races. This variability does not depend upon mutations having a random distribution with regard to dominance and recessiveness: it depends upon pairs of genes, most of which show at least a fair degree of dominance (one allelomorph having more than three times the effect of the other upon the heterozygote). In the case of these pairs of genes, it is usually the recessive allelomorph

which is infrequent ("abnormal") in the population and the dominant allelomorph which is frequent ("normal"). It will be noted that exactly these postulates are made in the current theory of heterosis, and that they are supported by considerations derived from modern work on mutation. In Wright's calculations concerning the distribution of gene frequencies, he arrives at a similar result, namely, that low frequencies of abnormal genes occur oftener than intermediate frequencies, and these abnormal genes are taken to be usually recessives. Our present conception also agrees with that of Wright in postulating that it is largely a matter of accident which particular loci happen to furnish most of the variance in a race (so far as ordinary quantitative morphological characters, of a sort necessarily dependent upon many genes, are concerned): thus two long-separated races, though having similar variance from a phenotypic standpoint, would owe this variance largely to different loci.

It may be explained at this point that the above-outlined conception does not run counter to the recent evidence obtained by Levit, that most pathological genetic traits in man are present in individuals heterozygous for the gene chiefly responsible, and that the latter gene therefore has some degree of dominance—enough to be important in pathology. This degree of dominance might, however, be as small as, for example, that of the more recessive allelomorph under the condition, $h = .1$. This would be considered, in our present paper, as corresponding to a fairly high degree of recessiveness. Nevertheless the relatively small amount of expression of the more recessive gene in the heterozygote in such a case would be sufficient to allow the gene to be discovered as a "dominant" in the case of many pathological traits. Hence these genes, though recessive in our sense, would still fall into the category found by Levit and referred to by him as "conditional dominants," and they would not violate the essential principle discovered by him: namely, that most of the genetically pathological individuals in

man are only heterozygous for the gene that causes the disturbance.

A further point which should be noted regarding our present conception is that it implies that the intraracial variability (that is, the inter-individual variability within a race) is comparable in magnitude or larger than the interracial variability, that is, than the difference between the mean of one race and that of another (even when we neglect the signs of the differences caused by each individual pair of genes and add all absolute differences together). If this were not so, then the partial suppression of the intraracial variability in the hybrid should not be able to compensate for the tendency to increased variability caused by the segregation of the interracial differences. An escape might be provided here by supposing the latter to depend on very numerous genes of individually smaller effect than the former; but, as we shall see later, this supposition is an unlikely one.

If the general conception above outlined be discarded, it might, alternatively, be postulated that most of the important gene frequencies fall along the diagonal "lines of minimal differences" of our tables, above discussed, that is, that the frequencies of the genes causing intraracial variability are very similar in the two races, locus for locus. In that case, we should not have to suppose an especially high concentration of genes exhibiting fairly good dominance, as compared with non-dominants. But in that case, it would be strange to find the phenotypic curve of one race widely different from that of the other race and practically not overlapping the latter—a condition found in the case of very many characters when groups derived from different major races of mankind (Negroes, Caucasians and Mongolians) are compared. We might, to be sure, suppose in this case that the variance in each race was due to very many genes, each having a relatively small effect, and that the difference between the means was brought about by the fact that in race 1, as compared with race 2, there was a great pre-

ponderance of slightly higher frequencies for just those genes which affected the character in a given direction. Such a preponderance of many genes working in the same direction within a given race could hardly have come about except by selection. But if selection could be thus effective in the case of so many loci at once, then it should also have been able to be effective in causing a very wide difference in gene frequency, in the case of an individual locus, that is, the frequencies of the genes in corresponding loci in the two races should no longer be so similar as they are here assumed to be. It is thus seen that this second hypothesis meets with a serious difficulty within itself.

The above hypothesis might be moderated to some extent by allowing the existence not only of frequencies ranging along the diagonal line in question, but also of some other frequencies, occurring in a more or less random fashion; there would then be *some* compensation of the increased variabilities caused by frequencies represented in the upper right-hand (and lower left-hand) corners of all the tables, by the decreased variabilities caused by frequencies represented in the upper left-hand regions of the tables for strongly recessive genes. But still, in order that hybrid variability be not greatly raised, it would have to be supposed that there was relatively little influence of the genes in the upper right-hand corner, that is, of genes in regard to which the two races were relatively homozygous for opposite allelomorphs. To avoid the dilemma regarding the cumulative effects of genes represented along the diagonal line, discussed in the preceding paragraph, it might then be supposed that the mean values of the two races did differ considerably as a result of homozygosity in opposite allelomorphs (*i.e.*, as a result of genes in the upper right-hand corners), but that these opposite allelomorphs were individually of very small influence and only collectively important. As was shown long ago by Wright, the variance in F_2 caused by the segregation of all such genes at once is, other

things being equal and assuming a constant difference between the two extreme races, inversely proportional to the square root of the number of genes involved (and therefore directly proportional to the square root of the amount of phenotypic influence of the individual members of such a group of multiple factors). But here again we could hardly explain the identity of direction of the great majority of the small genetic differences between the races, except by selection. The question would then arise, why should these genes differentiating the races be individually so small in their effects as to cause an almost negligible variance in F_2, while the genes causing the intraracial variabilities had so much larger effects? Is it sufficient here to say that selection is concerned mainly with very small mutations, while the intraracial variability is still largely unselected and depends mostly on larger mutations? Even if this were true, we should then be compelled to ascribe nearly all conspicuous race differences to selection.

In order to decide between these various possibilities, still more, to obtain a quantitative estimate of the degree to which one or another applies, new anthropological, zoological and botanical data should be obtained, gathered with a view to these genetic desiderata. For the reasons given in the first section, present anthropological data are decidedly inadequate (and in other organisms the problem seems to have been little touched). It is not even quite certain, as yet, that there is not usually some appreciable increase in the variability of hybrid races of man, as compared with the original races. To make sure of this, the data on the original races must be taken as nearly as possible for the same groups, or mixtures of groups, as those which entered into the composition of the hybrid races. Characters which, like weight and height, are considerably affected by environmental influences, should be avoided. Both those characters in regard to which the two races show a considerable difference of means and those in regard to which the means are

much alike should be investigated; both phenotypically overlapping and non-overlapping characters. Except in cases like that of the Norfolk Islanders, where breeding has almost certainly been random for many generations (so far as racial composition was concerned), exact pedigree studies must be made and then only those individuals which have genetically the same kind of mixture of the two races must be grouped together. It is also evident that cases must be avoided in which there is reason to believe that one of the "original" races was itself in a very considerable degree a mixture of two major races, especially if one of the latter is the same as, or akin to, the second "original" race. Thus it is evident, for example, that a race derived by crossing of the present Norfolk Islanders with whites would be "purer" than the present race of Norfolk Islanders. In the case of human races, this is a difficult condition to be sure of, and hence only the groups which are clearly of more extreme type can be used.

Much valuable light can also be thrown on the problems at issue if studies are made, not only of a group corresponding to the F_2 or random mixture, but also on F_1's and other types of descendants of a mixture. If most of the variance and most of the difference between the means of the original races were due to genes whose frequencies lie along the diagonal line of minimal differences, then the F_1 should have just about the same amount of variability as the original races and as the F_2. Otherwise the F_1 should be distinctly less variable; that is, any considerable scattering of the gene frequencies over parts of the table far from the diagonal should lead to a relatively decreased variability of F_1.

Legend to Tables B to G

Effects of genes of various frequencies (p) and degrees of dominance (h) in race crosses (p_1 and p_2 are frequencies of the same relatively recessive gene in first and second original races respectively). In each rectangle, lowermost figure shows difference in means of original races caused by this gene; middle figure—average variance of both original races taken together, caused by this gene; uppermost figure—increase of variance in the mixed race, relative to average variance of original races, caused by this gene.

TABLE B.	(h = 0.0)	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	p_1/p_2
		−.496 .0025 .010	−.479 .0096 .040	−.463 .0205 .090	−.429 .0336 .160	−.375 .0469 .250	−.288 .0576 .360	−.141 .0625 .490	.167 .0576 .640	1.097 .0385 .810	∞ 0 1.0	0
		0 .0050 0	−.091 .0121 .030	−.165 .0230 .080	−.188 .036 .150	−.169 .0494 .240	−.107 .0601 .350	.033 .0650 .480	.341 .0601 .630	1.288 .0410 .800	41.2 .0025 .990	.1
			0 .0192 0	−.026 .0301 .050	−.051 .0432 .120	−.049 .0565 .210	0 .0672 .320	.119 .0721 .450	.396 .0672 .600	1.193 .0481 .700	11.0 .0096 .960	.2
TABLE C.	(h = .1)											
.1	−.382 .00275 .028	0 .0055 0		0 .0410 0	−.008 .0541 .070	−.0003 .0674 .160	.033 .0781 .270	.130 .0830 .400	.350 .0781 .550	.955 .0590 .720	4.95 .0205 .910	.3
.2	−.421 .0095 .072	−.068 .01225 .044	0 .0190 0		0 .0672 0	.002 .0805 .090	.029 .0912 .200	.098 .0961 .330	.264 .0912 .480	.692 .0721 .650	2.72 .0336 .840	.4
.3	−.406 .0192 .132	−.133 .02195 .104	−.020 .0287 .060	0 .0384 0		0 .0938 0	.010 .105 .110	.053 .1094 .240	.168 .1045 .390	.465 .0854 .560	1.625 .0469 .750	.5
.4	−.375 .0304 .208	−.152 .03315 .180	−.037 .0399 .136	−.005 .0496 .076	0 .0608 0		0 .1152 0	.015 .1201 .130	.085 .1152 .230	.279 .0961 .460	1.000 .0576 .640	.6
.5	−.318 .04125 .300	−.126 .04395 .272	−.027 .05075 .228	.006 .06045 .168	.004 .07165 .092	0 .0825 0		0 .1250 0	.024 .1201 .150	.141 .1010 .320	.605 .0625 .510	.7
.6	−.225 .0496 .408	−.056 .05235 .380	.027 .0591 .336	.046 .0688 .276	.031 .0800 .200	.010 .09085 .108	0 .0992 0		0 .1152 0	.042 .0961 .170	.335 .0576 .360	.8
.7	−.064 .0528 .532	.094 .05555 .504	.156 .0623 .460	.144 .0720 .400	.104 .0832 .324	.054 .09405 .232	.016 .1024 .124	0 .1056 0		0 .0770 0	.143 .0385 .190	.9
.8	.270 .0479 .672	.422 .05065 .644	.439 .0574 .600	.367 .0671 .540	.267 .0783 .464	.167 .08915 .372	.082 .0975 .254	.025 .1007 .140	0 .0958 0		0 0 0	1.0
.9	1.283 .03155 .828	1.407 .0343 .800	1.236 .04105 .756	.954 .05075 .696	.680 .06195 .620	.449 .0729 .528	.270 .08115 .420	.135 .08435 .296	.042 .07945 .156	0 .0631 0		
1.0	∞ 0 1.000	32.4 .00275 .972	9.44 .0095 .928	4.43 .0192 .868	2.47 .0304 .792	1.501 .04125 .700	.930 .0496 .592	.567 .0528 .468	.318 .0479 .328	.139 .03155 .172	0 0 0	
p_2/p_1	0	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	

TABLE D.	(h = .2)	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	p_1/p_2
		−.227 .00375 .046	−.286 .0105 .104	−.285 .01915 .174	−.260 .0284 .256	−.206 .0369 .350	−.105 .0428 .456	.074 .04435 .574	.448 .0393 .704	1.592 .0254 .846	∞ 0 1.000	0
		0 .0075 0	−.038 .01425 .058	−.083 .0229 .128	−.088 .03215 .210	−.058 .04065 .304	.022 .04655 .410	.180 .0481 .528	.522 .04305 .658	1.536 .0291 .800	20.50 .00375 .954	.1
TABLE E.	(h = .3)		0 .0210 0	−.011 .02965 .070	−.015 .0389 .152	.006 .0474 .246	.065 .0533 .352	.200 .05485 .470	.481 .0498 .600	1.250 .03585 .742	7.155 .0105 .896	.2
.1	−.099 .00555 .064	0 .0111 0		0 .0383 0	.001 .04755 .082	.012 .05605 .176	.061 .06195 .282	.162 .0635 .400	.380 .05845 .530	.925 .0445 .672	3.62 .01915 .826	.3
.2	−.118 .0126 .136	−.019 .01815 .072	0 .0252 0		0 .0568 0	.007 .0653 .094	.038 .0712 .200	.108 .07275 .318	.264 .0677 .448	.646 .05375 .590	2.122 .0284 .744	.4
.3	−.123 .0203 .216	−.024 .02583 .152	−.003 .0329 .080	0 .0406 0		0 .0738 0	.012 .0797 .106	.053 .08125 .224	.162 .0762 .354	.425 .06225 .496	1.318 .0369 .650	.5
.4	−.090 .0277 .304	−.012 .03325 .240	.009 .0403 .168	.019 .0480 .088	0 .0554 0		0 .0856 0	.016 .08715 .118	.080 .0821 .248	.254 .06815 .390	.838 .0428 .544	.6
.5	−.027 .03375 .400	.033 .0393 .336	.054 .04635 .264	.024 .05405 .182	.008 .06145 .096	0 .0675 0		0 .0887 0	.021 .08365 .130	.128 .0697 .272	.516 .04435 .426	.7
.6	.090 .0373 .504	.141 .04285 .440	.110 .0499 .368	.073 .0576 .288	.038 .0650 .200	.010 .07105 .104	0 .0746 0		0 .0786 0	.039 .06465 .142	.290 .0393 .296	.8
.7	.317 .0371 .616	.296 .04265 .552	.247 .0497 .480	.177 .0574 .400	.108 .0648 .312	.052 .07085 .216	.015 .0744 .112	0 .0742 0		0 .0507 0	.129 .02535 .154	.9
.8	.740 .0318 .736	.657 .03735 .672	.522 .0444 .600	.376 .0521 .520	.254 .0595 .432	.150 .06555 .336	.072 .0691 .323	.020 .0689 .120	0 .0636 0		0 0 0	1.0
.9	2.100 .01995 .864	1.646 .0255 .800	1.206 .03255 .728	.854 .04025 .648	.584 .04768 .560	.380 .0537 .464	.227 .05725 .360	.114 .05705 .248	.033 .05175 .128	0 .0399 0		
1.0	∞ 0 1.000	11.94 .00555 .936	4.925 .0126 .864	2.72 .0203 .784	1.68 .0277 .696	1.082 .03375 .600	.708 .0373 .496	.442 .0371 .384	.254 .0318 .264	.108 .01995 .136	0 0 0	
p_2/p_1	0	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	

TABLE F.	(h = .4)	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	p_1/p_2
		.002 .0080 .082	.018 .01575 .168	.054 .02265 .258	.115 .02825 .352	.216 .0319 .450	.371 .03305 .658	.652 .03305 .552	1.228 .02535 .768	2.993 .0152 .882	∞ 0 1.000	0
		0 .0160 0	.007 .02375 .086	.028 .03065 .176	.067 .03625 .270	.134 .0399 .368	.250 .04105 .470	.446 .03905 .576	.820 .03335 .686	1.75 .0232 .800	7.21 .0080 .918	.1
TABLE G.	(h = .5)		0 .0315 .0	.008 .0384 .090	.030 .0440 .184	.077 .04765 .282	.158 .0488 .384	.296 .0468 .490	.551 .0411 .600	1.12 .03095 .714	3.20 .01575 .832	.2
.1	.059 .01125 .100	0 .0225 0		0 .0453 0	.008 .0509 .094	.035 .05455 .192	.090 .0557 .294	.188 .0537 .400	.367 .0480 .510	.749 .03785 .624	1.865 .02265 .742	.3
.2	.125 .0200 .200	.020 .03125 .100	0 .0400 0		0 .0565 0	.009 .06015 .098	.040 .0613 .200	.106 .0593 .306	.233 .0536 .416	.493 .04345 .530	1.20 .02825 .648	.4
.3	.215 .02625 .300	.067 .0375 .200	.0135 .04625 .100	0 .0525 0		0 .0638 0	.010 .06495 .102	.050 .06295 .208	.132 .05725 .318	.320 .0471 .432	.799 .0319 .550	.5
.4	.336 .0300 .400	.137 .04125 .300	.0500 .0500 .200	.011 .05625 .100	0 .0600 0		0 .0661 0	.011 .0641 .106	.064 .0584 .216	.189 .04825 .330	.534 .03395 .448	.6
.5	.500 .03125 .500	.235 .0425 .400	.110 .05125 .300	.043 .0575 .200	.01 .06125 .100	0 .0625 0		0 .0621 0	.018 .0564 .110	.099 .04625 .224	.348 .03105 .342	.7
.6	.750 .0300 .600	.378 .04125 .500	.200 .0500 .400	.100 .05625 .300	.042 .0600 .200	.010 .06125 .100	0 .0600 0		0 .0507 0	.030 .04055 .114	.200 .02535 .232	.8
.7	1.166 .02625 .700	.600 .0375 .600	.338 .04625 .500	.190 .0525 .400	.100 .05625 .300	.043 .0575 .200	.011 .05625 .100	0 .0525 0		0 .0304 0	.092 .0152 .118	.9
.8	2.000 .0200 .800	.981 .03125 .700	.563 .0400 .600	.338 .04625 .500	.200 .0500 .400	.110 .05125 .300	.050 .050 .200	.013 .04625 .100	0 .0400 0		0 0 0	1.0
.9	4.500 .01125 .900	1.779 .0225 .800	.981 .03125 .700	.600 .0375 .600	.378 .04125 .500	.235 .0425 .400	.137 .04125 .300	.067 .0375 .200	.020 .03125 .100	0 .0225 0		
1.0	∞ 0 1.000	4.50 .01125 .900	2.00 .0200 .800	1.166 .02625 .700	.750 .0300 .600	.500 .03125 .500	.333 .0300 .400	.216 .02625 .300	.125 .0200 .200	.058 .01125 .100	0 0 0	
p_2/p_1	0	.1	.2	.3	.4	.5	.6	.7	.8	.9	1.0	

Wherever possible, data on relatives, particularly on groups of brothers and of sisters, should be obtained, in the original races, in F_1, and especially in F_2 and, separately, in later generations produced by random interbreeding. For if the conception above advanced as the more probable is correct, namely, that a considerable amount of the original intraracial variability is suppressed in the mixed race and replaced by important interracial differences, now segregating, then it will be seen that the correlation between relatives will be considerably less in F_2 than it was in the original races, for the segregation of the interracial differences will tend to manifest itself within the individual F_2 families about as much as in the F_2 population as a whole, while the original intraracial differences, which of course still have a tendency to be grouped in families, will be diminished in expression. On the other hand, in all later generations than the F_2, of a random-breeding mixture, while the familial distribution of the original intraracial variations remains the same as in F_2, the segregating interracial differences will tend to assume a familial form also, so that intra-familial correlation will again increase above that of the F_2. (In the absence of any tendency to inbreeding it will be as high in F_3 as in any later generation, *i.e.*, it will remain constant after F_3.)

On the other hand, in so far as the relatively homozygous opposite allelomorphs of the two original races had very small effects individually (even though large effects collectively), they would have little effect on the variability within the family in F_2, just as they would have little effect within the whole F_2 population and within the mixed race as a whole. Hence if a markedly decreased fraternal correlation is really found in F_2 it will speak for the existence of opposite, relatively homozygous allelomorphs which individually have a relatively large effect, just as postulated in the theory which we favor. And then it would be necessary to conclude that,

in compensation for this, there was also a large group of low-frequency recessive genes, of the type which decrease the variability of the hybrids and cause intraracial variability in the original race.

We do not, however, wish to insist here upon the high probability of the particular conception which we tend to favor, but rather to show that there are in fact various Mendelian interpretations of that lack of increase of variability in hybrid populations which has been claimed to exist by some anthropologists, and that a decision can probably be made from among these various possible explanations by means of data of a sort that should be obtainable. Further tables (and also, so far as possible, general formulae) should be constructed, showing the influence of the different possible sorts of genetic situations on the familial correlation or, what amounts to the same thing, on the intra-familial variance as compared with the whole intra-populational variance, in the original races and in F_1, F_2 and F_3. The data obtained by observation should then be compared with the expectations as given by these latter tables and formulae and by the tables presented in our present paper. It will probably be found that the observations fit in only with a fairly limited set of presupposed genetic conditions, when all the values mentioned are taken into consideration at once. The answers thus obtained to the problems here raised would be of interest because of the various implications which they would have in connection with modern questions concerning dominance, concerning the relative numbers of genes having different frequencies, and concerning differential multiplication, selection, mutation and evolution problems in general. As yet, such problems have hardly been attacked at all by anthropologists or even by systematic biologists working on other organisms. It would seem that they may now begin to obtain some data on these more underlying questions.

Before closing, a few words should be said concerning the bearing of the matters dealt with in this paper upon

the question of the fitness of individuals of mixed race. Most anthropologists and geneticists agree that the first generation offspring from race crosses are, on the whole, as advantageously equipped and have no greater tendency to disharmonies of structure and function than the members of the original populations. We leave out of account here the matter of special adaptations to particular climates and modes of life, such as the protective skin coloration of the Negro in regions of intense sunlight, or the short intestine of the Eskimo with its higher adaptation for a carnivorous diet, and refer only to characteristics of more general utility. This procedure is especially legitimate in the case of man to-day, in view of his rapidly increasing ability to compensate by artificial means for the particular local conditions of his environment. In contrast to the first generation, however, it has been claimed by some writers on the subject of race that the recombinations formed in later generations were apt to be "disharmonious," and so in general less fit. It may be granted that this stricture does undoubtedly apply to the products of crosses between widely different species, having in fact been directly proved in many crosses of plant species. It would not, however, be expected to apply to race crosses in so far as the characters in these crosses conform to the rule above discussed, *i.e.*, in so far as they show little or no greater variability in the mixed than in the original races, and the same kind of variability. For if the mean of the mixed race is as fit as that of the original races, and the variation from the mean is no greater than in the latter, the variants also should on the average be as fit in the mixed race as in the original races, and this would apply not only to individual characters but to recombinations of them.

The above conclusion remains valid, no matter which proposed interpretation of the unincreased variability of the mixed race be adopted. According to our first and preferred interpretation, a part of the original intraracial variability has been suppressed and the place of

this has been taken, in part at least, by variation due to the segregation of interracial differences. In so far as the latter variation is little or no greater than that which it replaces, it should not be more harmful. On the contrary, it might be expected, from a qualitative point of view, to be less harmful, since it involves the segregation of genes both allelomorphs of which have had a chance to be subjected to the test of prolonged natural selection and which should therefore be more innocuous than the relatively unselected random and sporadic individual variations that they replace. This does not exclude the possibility of incompatibilities in special cases—especially those of a chemical kind not demonstrable by our morphological measures—but there might be a still higher frequency of such disharmonies arising in intraracial breeding, when all characters were taken into account at once. And the fact should also be taken into consideration that the establishment of characters in one race which were disharmonious with those of the other race should on the whole be effectively hindered by natural selection, in the cases of races like the major human races, in which, by occasional direct or indirect crossing, there occurs a slight infiltration of genes from one race to the other. For the continual presence of the latter genes, even though in only a small number of individuals, creates a situation in which the establishment of other genes, incompatible with these, will be prevented by selection (except in the rare cases where these other genes have a more than compensatory advantage of some other sort).

If now we turn to the second alternative interpretation, that most of the variation in the mixed race is caused by the same genes as those which produce the variation in the original races (these genes in the two original races having had only slight differences in frequency), then again we must conclude that the variation among individuals of the mixed race would not tend to be more harmful than that in the original races, for then the variation in the mixed race would be of practically the same

sort, both quantitatively and qualitatively, as that in the original races. This conclusion would apply not only to the variations caused by each pair of genes considered separately, but also to that caused by the process of recombination between genes at different loci. Hence we see that both of the two possible interpretations would be expected to give results that are in harmony with the experience of history, *viz.*, that mixed races of man are not *per se* "inferior."

The author hereby acknowledges with thanks his appreciation of the generous assistance given him by the statistical department of the Medicogenetical Institute, Moscow, in rechecking the computations of the tables.

RÉSUMÉ

Anthropological data, indicating that mixed races formed by hybridization between the major known races have on the whole little or no more variability, and perhaps in some cases even less, than the original races, are in no wise out of accord with the principles of modern genetics, based on Mendelism. On any Mendelian interpretation, however, the unincreased variability can not (as sometimes stated) be related to the number of generations of breeding of the mixed race, but must be as evident in F_2 as later.

Several Mendelian interpretations are possible. What appears to us the most plausible involves the postulate that most of the intraracial variation of the original races is caused by relatively recessive genes that individually (at each given locus) are rather rare, but taken together are numerous, and the further postulate that the causation of this variation is for the most part referable to different loci in the different major races, even though a variation of a similar phenotypic kind results. On this interpretation a part of the intraracial variance of the original races would be suppressed in the mixed race (see simple arithmetical example in section 2), its place being taken by the segregation of interracial differences. Since

these two causes of change in the variance would rarely compensate each other exactly, the mixed race may show either some increase or decrease of variance, as compared with the original races. The fact that the increase or decrease was small would then indicate that the inter-individual differences within a race are of comparable magnitude or greater than the interracial differences between the original major races. In view of the latter conclusion (especially when taken in connection with the consideration that the racial peculiarities have had more chance to be sifted out by selection than the individual ones) it is not to be expected that the recombinations produced by various kinds of racial mixture will have any greater tendency to be disadvantageous than those arising by the breeding together of individuals of the same original race.

An alternative Mendelian interpretation would postulate that the original intraracial differences were due mainly to genes occupying the same loci in the two major races and having very similar but not quite identical frequencies in these races. The difference between the means of the two races would then depend either on the cumulative effect of these frequency differences, present in numerous loci and tending to act in the same phenotypic direction, or else upon other genes, individually very small in their effect, but collectively very numerous, in regard to which the two races were relatively homozygous for opposite allelomorphs. Either of these latter postulates, however, meets with serious difficulties, in explaining how such a condition could have arisen by selection, without greater differences having arisen in the frequencies of the genes causing intraracial variation. It is to be noted that on this interpretation, also, interracial recombinations should on the average have no more tendency to be disadvantageous than intraracial ones.

A table (A) is given, showing quantitatively the effects which would be produced on the variance of a race by the presence of alternative allelomorphs having different fre-

quencies and degrees of dominance. Further tables (B to G) show how much (for any given locus) the variance of the mixed race would be decreased or increased relatively to that of the original races, under various possible conditions of gene frequency and dominance in each of the two races crossed. The effect of the assumed conditions on the difference between the means of the races is also given. These tables are of use in judging the genetic meaning of a given body of data on race and hybrid variability.

The requirements are stated which should be met by any future body of data dealing with the problem at issue, in order that a qualitative and quantitative decision may be reached between the various possible Mendelian interpretations stated. It is shown that separate data concerning F_1 and F_2 and later generations are needed and data on intra-familial correlation in F_1, F_2 and later generations, as compared with that in the original races; the ways in which such data would bear upon the problem are pointed out.

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COMPARATIVE STUDY OF FOUR HIGH TUMOR LINES OF MICE

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COMMUNICATIONS have been published on the breeding behavior and tumor incidence of several strains of mice which are being used for experimental purposes at this laboratory. In this article we wish to make comparative studies of the data.

STOCKS TO BE COMPARED

(1) The "D" strain. The animals of this strain are descended from the Little dilute brown race. They have been inbred by brother-to-sister matings since 1909. The diet which this strain received consisted of hemp and canary seed, cod liver oil puppy cakes and milk. Lettuce or cabbage was given once a week. The breeding and tumor observations have previously been reported for this stock by Murray (4).

(2) The "A" strain. This strain originated from the Bagg albino stock, being inbred since 1912. Prior to 1932 the food which the animals received was a mixture of rolled oats, powdered milk, powdered meat scraps and salt; cod liver oil puppy cakes and water, with lettuce or cabbage being added once a week. Since 1932 the animals have had Purina Fox Chow and water. For simplicity, the line of the A stock which received the rolled oats diet shall be termed the A–RO line and the other the A–FC line.

The number of animals which developed spontaneous mammary gland tumors in two other lines of the A stock has been reported by Strong (5). The number of individuals dying from other causes than cancer was not given. Thus the data observed by Strong can not be compared with that recorded in this report.

The breeding behavior and the tumor incidence depending upon the diet which the animals received is now in press (1, 3).

(3) The C_3H or "Z" Strain. A black agouti stock which has been inbred since 1918. The mice were raised on the Purina food. A description of the breeding behavior and tumor observation is being reported (2).

BREEDING DATA ACCORDING TO THE AGE OF MOTHER

The average age of the mothers at the birth of the first litters in the various stocks was: D, 117 days; A–RO, 91 days; A–FC, 81 days; and C_3H, 92 days. The difference between the two lines of the A stock is due mainly to a few females which received the rolled oats diet having their first litters at a very late age. If these are excluded the average for the remainder is 84 days instead of 91 days. The average for the D stock is older for the same reason.

The mean litter sizes are given in Table 1 to demonstrate the effect the age of the mothers may have upon the average number of young born per litter. Fig. 1 is a graphic representation of the data.

TABLE 1

MEAN LITTER SIZE ACCORDING TO THE AGE OF THE MOTHERS

	D Stock		A–RO Stock		A–FC Stock		Z or C_3H Stock	
	No. of litters	Mean	No. of litters	Mean	No. of litters	Mean	No. of litters	Mean
51– 80	516	4.2 ± 0.05	141	4.7 + 0.11	135	5.1 ± 0.1	91	4.1 ± 0.12
81–110	1,299	5.0 ± 0.04	143	5.1 ± 0.11	120	5.5 ± 0.1	110	5.7 ± 0.10
111–140	1,024	5.4 ± 0.04	116	5.4 ± 0.12	136	6.5 ± 0.1	112	5.4 ± 0.08
141–170	976	5.6 ± 0.04	109	5.2 ± 0.14	126	6.6 ± 0.1	108	5.9 ± 0.11
171–200	761	5.5 ± 0.05	96	5.3 ± 0.14	125	6.2 ± 0.1	76	5.8 ± 0.14
201–230	693	5.4 ± 0.06	104	5.0 ± 0.12	123	6.2 ± 0.1	79	5.9 ± 0.14
231–260	600	5.0 ± 0.06	76	4.4 ± 0.16	124	6.2 ± 0.1	66	5.4 ± 0.13
261–290	434	5.4 ± 0.06	65	4.5 ± 0.17	100	5.5 ± 0.1	45	5.2 ± 0.18
291–320	285	5.1 ± 0.08	45	3.6 ± 0.23	81	5.3 ± 0.2	38	4.8 ± 0.19
321–350	196	4.6 ± 0.10	30	3.9 ± 0.20	71	4.3 ± 0.1	21	4.7 ± 0.29
351–380	104	4.3 ± 0.28	18	3.2 ± 0.28	51	3.9 ± 0.2	12	4.3 ± 0.26
381–410	50	4.3 ± 0.34	8	2.8 ± 0.58	25	3.6 ± 0.3	6	4.2
411 +	33	3.5 ± 0.25	7	3.0 ± 0.19	18	3.3 ± 0.4	4	4.0
Total	**6,971**	**5.2 ± 0.02**	**958**	**4.8 ± 0.04**	**1,235**	**5.7 ± 0.04**	**768**	**5.2 ± 0.04**

There is a gradual increase in the size of the litters born to the dilute brown females until they are over 111 days

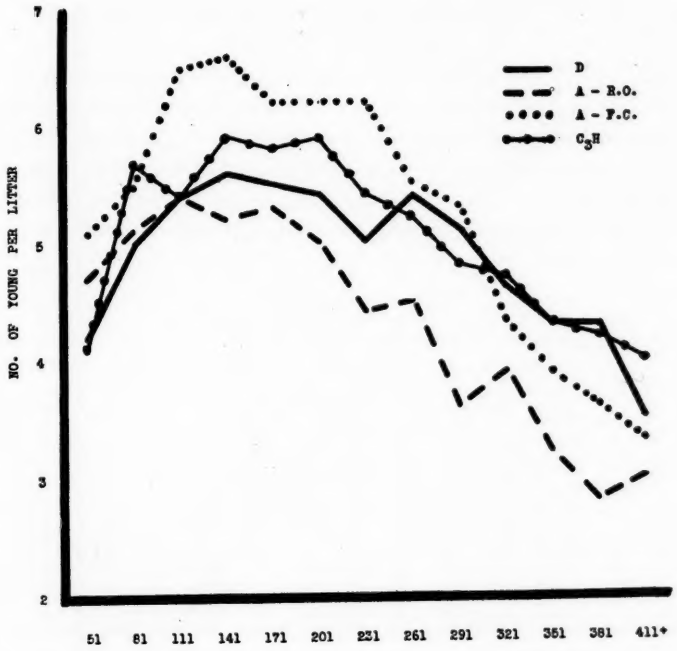

FIG. 1. Age of mother. Compares the mean litter size according to the stock of mice.

old. From this age to 230 days is the period of maximum production. The decrease in litter size probably starts after the mothers have reached 231 days of age, regardless of the increase in average litter during the period from 261–290 days.

In the A–RO line the largest litters were born to mothers between the ages of 81 to 230 days. The A–FC line females were one month older at the period when maximum size litters were produced and 261 days old before the average litter size started to decline.

A gradual diminishing mean litter size was observed in the C_3H strain after the 230 day age period.

The mean for all the litters of each stock was as follows: D, 5.2; A–RO, 4.8; A–FC, 5.7; and C_3H, 5.2.

EFFECT OF THE AGE OF MOTHER ON THE PERCENTAGE OF YOUNG WEANED

Each of the strains under consideration showed an increase in the percentage of the young raised to weaning age (28 to 30 days) until the mothers average from 111 to 170 days. For every race the period showing the largest proportion of young weaned represented approximately the month during which the second litters were born to the mothers.

TABLE 2

PERCENTAGE OF YOUNG WEANED DEPENDING ON THE AGE OF THE MOTHERS

Age of mother	D Stock		A–RO Stock		A–FC Stock		Z or C_3H Stock	
	No. born	Per cent. weaned	No. born	Per cent. weaned	No. born	Per cent. weaned	No. born	Per cent. weaned
51– 80	2,156	52.5 ± 0.7	656	31.7 ± 1.2	692	70.1 ± 1.2	371	73.0 ± 1.6
81–110	6,490	55.6 ± 0.4	734	39.1 ± 1.2	656	77.4 ± 1.4	521	74.1 ± 1.3
111–140	5,565	56.9 ± 0.5	631	41.2 ± 1.3	889	82.3 ± 0.9	601	71.9 ± 1.2
141–170	5,447	58.8 ± 0.5	561	47.2 ± 1.4	831	82.8 ± 0.9	633	82.0 ± 1.0
171–200	4,188	54.5 ± 0.5	506	41.1 ± 1.5	779	76.1 ± 1.1	438	73.1 ± 1.4
201–230	3,716	52.2 ± 0.6	522	46.7 ± 1.5	764	79.2 ± 1.0	465	71.0 ± 1.4
231–260	3,028	45.7 ± 0.6	336	42.3 ± 1.8	763	73.0 ± 1.0	358	60.9 ± 1.7
261–290	2,242	37.2 ± 0.7	291	38.8 ± 1.9	549	67.2 ± 1.4	233	45.9 ± 2.2
291–320	1,461	29.7 ± 0.8	162	35.8 ± 2.5	433	75.3 ± 1.4	184	44.6 ± 2.4
321–350	905	31.5 ± 1.0	117	43.6 ± 3.1	307	59.0 ± 1.9	98	51.3 ± 3.4
351–380	454	21.4 ± 1.3	58	39.7 ± 4.3	198	43.4 ± 2.4	52	32.7 ± 4.4
381–410	222	14.4 ± 1.6	30	13.3 ± 4.3	91	36.3 ± 3.4	25	12.0 ± 4.4
411 +	116	12.1 ± 2.0	21	28.6 ± 6.7	60	26.7 ± 3.8	16	0.0
Total	**35,990**	**51.2 ± 0.2**	**4,625**	**40.4 ± 0.5**	**7,012**	**73.9 ± 0.4**	**3,995**	**68.5 ± 0.5**

After the mothers had attained an average of 230 days there was a decline in the weaning percentage in each stock. This decrease was less apparent for the A–RO line, apparently due to the poor record this line had as compared with the other strains.

Fifty-one per cent. of the young born in the D stock were weaned; 40 per cent. of the A–RO; 74 per cent. of the A–FC; and 69 per cent. of the C_3H.

MAMMARY TUMOR INCIDENCE

The number of spontaneous mammary gland tumors which was observed in each stock according to monthly periods is recorded in Table 3. Curves for the data in bimonthly periods are given in Fig. 4.

The modes for the D and the C_3H strains occurred during the tenth month. Both lines of the A stock showed

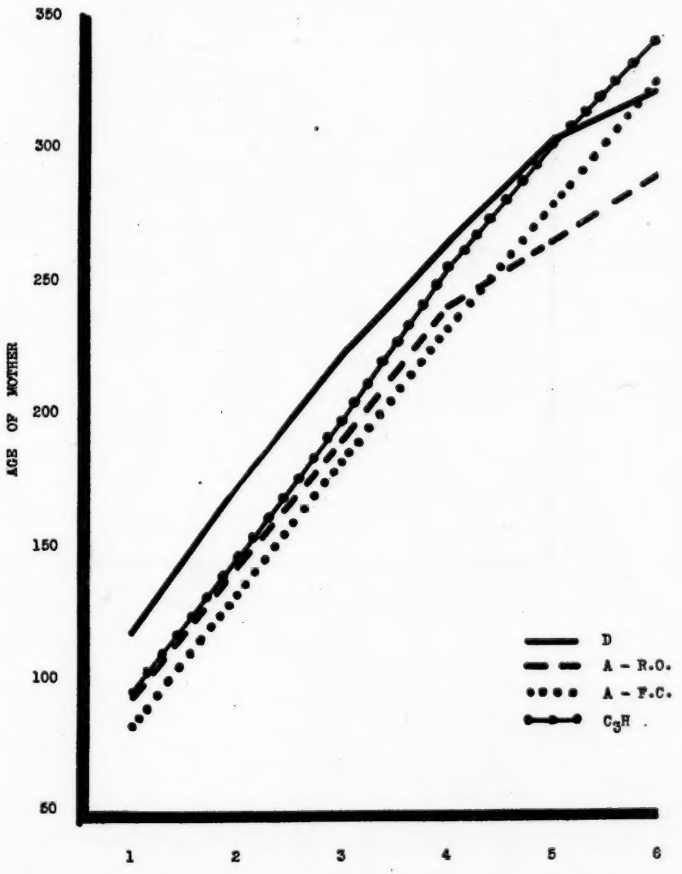

FIG. 2. Order of litter. Shows average age of the mother according to stock at the birth of successive litters.

a bimodal tendency, the A–RO line during the ninth and the thirteenth months and the A–FC line for the ninth and the twelfth months. The bimodal characteristics are eliminated if the data are grouped according to bimonthly age periods (Fig. 4).

The youngest animal to be observed with a tumor was a D stock individual and the oldest was from the A–RO

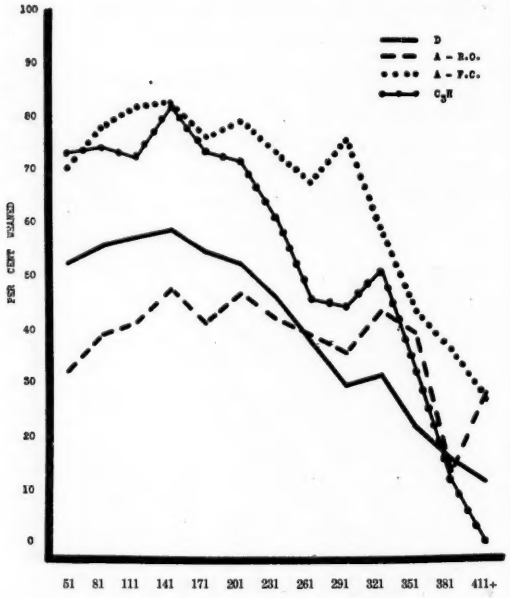

FIG. 3. Age in bimonthly periods.

TABLE 3

THE NUMBER OF ANIMALS DEVELOPING CANCER EACH MONTH AND THE PER CENT. OF THE TOTAL

Age in months	D Stock		A–RO Stock		A–FC Stock		Z or C3H Stock	
	No. ca.	Per cent. of total	No. ca.	Per cent. of total	No. ca.	Per cent. of total	No. ca.	Per cent. of total
3.5	4	0.3	0	0.0	0	0.0	0	0.0
4.5	9	0.7	2	0.7	0	0.0	0	0.0
5.5	25	1.9	1	0.4	2	0.8	1	0.6
6.5	67	5.1	7	2.6	2	2.6	11	7.1
7.5	95	7.2	13	4.8	10	4.8	4	2.6
8.5	170	12.9	14	5.2	20	5.2	13	8.3
9.5	198	15.0	35	13.0	36	13.0	19	12.2
10.5	208	15.8	31	11.5	34	11.5	27	17.3
11.5	190	14.4	31	11.5	29	11.3	23	14.7
12.5	129	9.8	28	10.4	39	15.2	18	11.5
13.5	86	6.5	36	13.4	26	10.1	11	7.1
14.5	48	3.6	18	6.7	27	10.5	10	6.4
15.5	41	3.1	19	7.1	11	4.3	2	1.3
16.5	25	1.9	16	6.0	9	3.5	8	5.1
17.5	13	1.0	7	2.6	1	0.4	6	3.9
18.5	5	0.4	5	1.9	6	2.3	2	1.3
19.5	3	0.2	1	0.4	3	1.2	1	0.6
20.5	0	0.0	3	1.1	1	0.4	0	0.0
21.5	2	0.2	0	0.0	1	0.4	0	0.0
22.5	0	0.0	1	0.4	0	0.0	0	0.0
23.5	0	0.0	1	0.4	0	0.0	0	0.0
Total ..	1,318	...	269	...	257	...	156	...

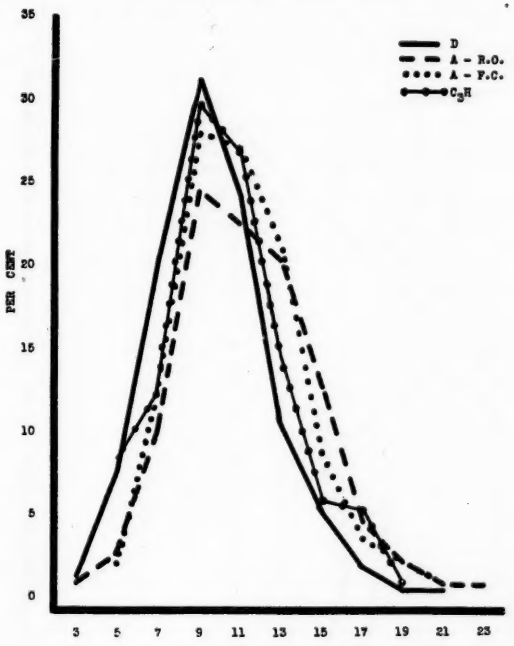

FIG. 4. Age of mother.

strain. The C_3H race showed the least variation in extreme age groups; the A–RO stock the greatest.

The mean age at the recording of tumors in the cancerous animals and at death of the non-tumorous individuals is summarized in Table 4. The mean tumor age for the

TABLE 4
MEAN AGE AT DEATH OF NON-CANCEROUS AND CANCEROUS ANIMALS ACCORDING TO STRAINS

Stock	No. non-ca.	Mean age non-cancerous	No. cancer	Mean age cancerous
D	934	9.5 ± 0.06	1318	10.6 ± 0.05
A–RO	152	9.1 ± 0.19	269	12.3 ± 0.13
A–FC	35	9.6 ± 0.47	257	11.5 ± 0.12
Z or C_3H	44	8.8 ± 0.19	156	10.7 ± 0.16

D and the C_3H strains are 10.6 and 10.7 months, respectively. The FC line mice of the A stock have their tumors at an earlier age than do the individuals of the

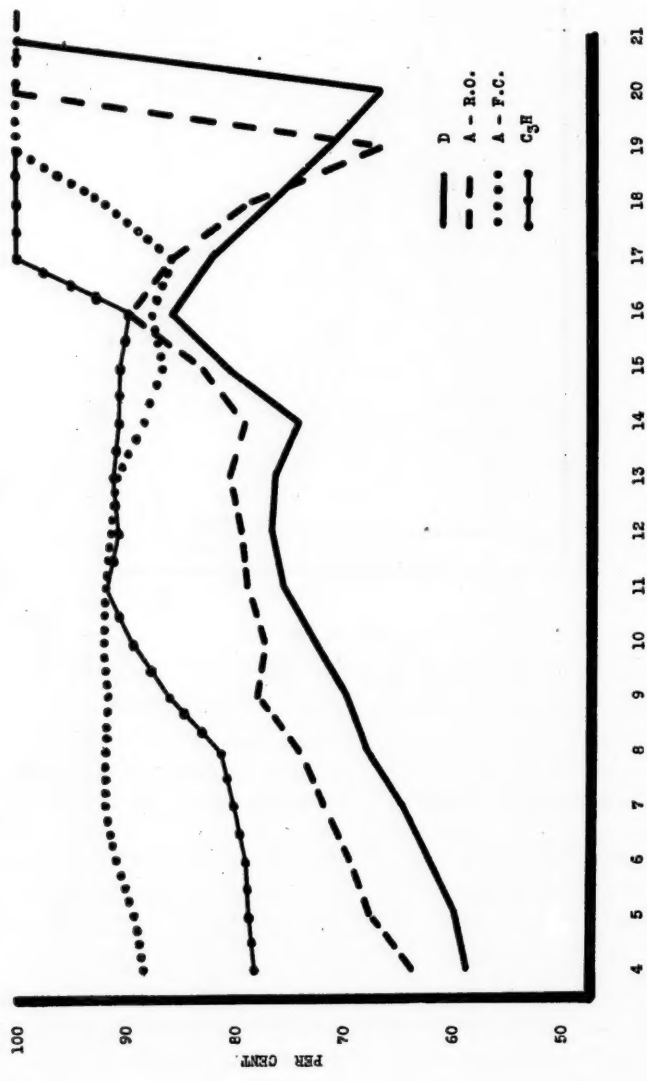

FIG. 5. Age in months. Compares the percentage of animals living to each age period or longer to develop cancer according to stock.

RO line (11.5 and 12.3 months). The age at death of the non-cancerous mice varied from 8.8 months for the C_3H strain to 9.6 for the A–FC line.

TABLE 5

THE NUMBER OF ANIMALS LIVING TO THE BEGINNING OF EACH MONTHLY AGE PERIOD AND THE NUMBER AND THE PER CENT. OF WHICH DEVELOPED MAMMARY GLAND CANCER

Age in months	D Stock			A–RO Stock			A–FC Stock			Z or C_3H Stock		
	Living	Died ca.	Per cent ca.	Living	Died ca.	Per cent. ca	Living	Died ca.	Per cent ca.	Living	Died ca.	Per cent ca.
3.5	2,251	1,318	58.6	...	...	....	...	...	....	...	...	....
4.5	2,239	1,314	58.7	421	269	63.9	292	257	88.0	200	156	78.0
5.5	2,187	1,305	59.7	395	267	67.6	289	257	88.9	199	156	78.4
6.5	2,066	1,280	62.0	383	266	69.5	281	255	90.7	197	155	78.7
7.5	1,873	1,213	64.8	360	259	71.9	276	253	91.7	180	144	80.0
8.5	1,651	1,118	67.7	331	246	74.3	265	243	91.7	172	140	81.4
9.5	1,363	948	69.6	298	232	77.9	244	223	91.4	148	127	85.8
10.5	1,033	750	72.6	256	197	77.0	204	187	91.7	121	108	89.3
11.5	718	542	75.5	211	166	78.7	167	153	91.6	88	81	92.0
12.5	460	352	76.5	171	135	79.0	136	124	91.2	64	58	90.6
13.5	294	223	76.0	133	107	80.5	94	85	90.4	44	40	90.9
14.5	185	137	74.1	90	71	78.9	67	59	88.1	32	29	90.6
15.5	111	89	80.2	64	53	82.8	37	32	86.5	21	19	90.5
16.5	56	48	85.7	38	34	89.5	24	21	87.5	19	17	89.5
17.5	28	23	82.1	21	18	85.7	14	12	85.7	9	9	100.0
18.5	13	10	76.9	14	11	78.6	12	11	91.7	3	3	100.0
19.5	7	5	71.4	9	6	66.7	5	5	100.0	1	1	100.0
20.5	3	2	66.7	5	5	100.0	2	2	100.0	...	...	....
21.5	2	2	100.0	2	2	100.0	1	1	100.0	...	...	....
22.5	....	....	....	2	2	100.0	...	...	....	...	...	....
23.5	....	....	....	1	1	100.0	...	...	....	...	...	....
Total	**2,251**	**1,318**	**58.6**	**421**	**269**	**63.9**	**292**	**257**	**88.0**	**200**	**156**	**78.0**

The albino line which received the Fox Chow diet showed the greatest percentage of cancerous animals observed before the eleventh month. Eighty-nine per cent. living five months or longer ultimately developed tumors (78 per cent. for the C_3H stock). After the eleventh month the C_3H strain gave the highest tumor incidence. This difference was probably due to the fact that the non-tumorous mice of the C_3H stock died at an earlier average age than in the A–FC line.

Of interest are the curves for the D stock and the A–RO line which showed marked similarity. At the fourth month the difference in tumor incidence was 5.2 per cent. and at the peak of the curve, which occurred at the sixteenth month, the variation was 3.8 per cent. After this period both curves showed a gradual decline in the percentage of cancerous individuals, which was followed by an increase.

The tumor incidence for the A–FC line and the C_3H strain differed considerably from the other two strains. The incidence in the D strain at the third month, the period when the first tumor was observed, was 58.6 per cent., which increased to 85.7 per cent. at the sixteenth month. At the fourth and sixteenth months the incidence in the A–RO line was 63.9 per cent. and 89.5 per cent., respectively. After this latter age period both strains showed a reduction in the proportion of cancerous animals. As the oldest animals of each strain died cancerous, the percentages ultimately increased to 100 per cent. —observations made on very small numbers. Eighty-nine per cent. of the A–FC line living longer than four months were cancerous. By the seventh month the proportion had increased to 91.7 per cent. and remained practically unchanged until the thirteenth month. From this period there was a decline until the sixteenth month (85.7 per cent.) and then an increase. Mammary gland tumors developed in 78.4 per cent. of the C_3H animals which lived to be five months or older. This percentage increased to 92 per cent. at the eleventh month and declined from this point to 89.5 per cent. at the sixteenth month.

Relationship Between Breeding Behavior and Tumor Incidence

A gradual diminishing in the mean litter size was noted in the D, A–RO and C_3H strains after the females had reached 231 to 260 days old. This took place in the A–FC line after 261 to 290 days. The proportion of young raised to weaning age in all strains was reduced after the mothers became 230 days old or older.

Thus, there is probably no correlation between the breeding behavior, as determined by litter size and mortality before weaning, and the proportion of animals developing mammary gland tumors.

There is, however, a direct relationship between the percentage of young weaned for any strain and the intervals between the birth of subsequent litters. That is,

strains which gave a high weaning percentage have a longer time between litters than a strain which showed a high mortality before weaning. This relationship is evidently due to the fact that a strain which had a poor record for weaning also would have a larger number of mothers which lost their entire litter at birth or a short time afterwards. These females would be returned to the breeding pen and would, on the average, produce a subsequent litter in a shorter interval than would animals from a strain which raised part or all of their litters more often.

The average age of the mothers at the birth of their first litters also probably has no influence on the mean age at the development of tumors. The D strain had the earliest mean tumor age and yet the mothers were considerably older when they gave birth to their first litters.

If there is a relationship between the breeding behavior and tumor incidence of a strain of mice, it is evident that our technique has not been developed sufficiently to detect such a connection.

Conclusion

There is probably no direct correlation between the mammary gland tumor incidence of a strain of mice and the breeding behavior, as expressed in litter size and weaning percentage according to the age of the females.

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LITTERS FROM FERRETS IN JANUARY INDUCED BY INCREASED EXPOSURES TO LIGHT AFTER NIGHTFALL[1,2]

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BISSONNETTE (1932) succeeded in inducing female ferrets to come into oestrus and mate with males in November and December by reversing their light cycles. But he found that, while males were caused to undergo increased germ-cell activity and complete activation of the accessory sex organs, such as the epididymis, they did not produce or ejaculate sperms under the conditions of the original experiment. Such matings merely rendered the females pseudopregnant.

At that time he thought that males were less susceptible than females to modification of their sexual and breeding cycles by changes in their light cycles. But, by modifying the method of increasing the length of day for males to a more gradual one, complete spermatogenesis with copious ejaculations of sperms was obtained in November, December and January in matings with females brought into oestrus by similar experimental lighting (Bissonnette, 1935 a, b, c, 1936).

These matings, however, did not produce young but only pseudopregnancy in the females, which were mated three or four times each within thirty to thirty-four hours with these experimentally activated males. At the time it was thought that the repeated matings would insure conception by obtaining ovulation and the presence of abundance of sperms in the reproductive tracts of the females. But apparently the added precautions defeated the end in view; for only pseudopregnancies occurred.

[1] Aided by grants from the National Research Council, Committee for Research in Problems of Sex, 1935–36.

[2] "Modification of Mammalian Sexual Cycles." VI.

In ferrets, ovulation occurs only after copulation, usually between thirty-four and thirty-six hours after the beginning of the period during which the pair remain fastened together. It does not occur spontaneously.

In the light of the studies of Foster and Hisaw (1935), and Witschi and Pfeiffer (1935) and others, it now seems probable that the repeated orgasms of these copulations, within the latent period before ovulation, caused luteinization of the follicles without ovulation. It has been shown that in the rabbit, and, therefore, probably in the ferret also, ovulation occurs when follicles, brought to maturity by the follicle-stimulating hormone of the anterior pituitary, come under the influence of increased amounts of the luteinizing hormone released from this lobe by the orgasm. This latter hormone is usually released in proper amounts to cause ovulation and subsequent corpus luteum formation by an orgasm induced by copulation or by artificial means. Therefore, it seems probable that the repeated copulations in so short a time freed the luteinizing hormone in such amounts as to cause luteinization without ovulation, and therefore prevented fertilization of the ova that would normally have been released by one copulation.

This hypothesis is also consistent with the fact, noted in some of Bissonnette's previous experiments, that even two copulations in about twenty-four hours caused ovulation from some follicles, with corpus luteum formation and fertilization of the eggs so ovulated, and also corpus luteum formation without ovulation in some other follicles in the same ovaries. This produced corpora lutea with ova still in their centers. At the time of these previous experiments it was not realized that ovulation depended on the liberation of luteinizing hormone and that excess of this hormone would luteinize follicles without ovulation, and even prevent ovulation.

The experiments to be described here repeated the previous ones with only one copulation for each female,

in light-induced oestrus, with a male in complete spermatogenesis similarly induced.

Success followed this method in the case of two females mated on November 29 and December 3. Both were pregnant; but the one mated in November died on January 5 from some wasting disease like tuberculosis. Her uterus was about three quarters of an inch in diameter throughout and foetuses were undergoing resorption before she died. The other female gave birth to six young on the afternoon or night of January 14, within the twenty hours succeeding the termination of the forty-second day after coition.

These young were born under winter conditions in a basement room with open windows in which water did not quite freeze, in a cage lined with zinc or galvanized iron and with scanty shavings for a bed and some excelsior shavings to cover them. Five of the six young died before January 20 from cold and exposure, when they were pushed away in the shavings or on the bare metal and the heat from the mother did not reach them. The sixth one lived until the night of February 6, when it died. None of the young were eaten by the female or killed by her. She was left in the metal-lined box until January 20 for fear that disturbing her would lead her to kill and eat her young. On that date, however, she was moved to a wooden box with plenty of shavings and paper towels for a nest.

The fate of these young suggests conditions such that ferrets having young out of their normal breeding season for this hemisphere meet with environmental factors which have selective action by eliminating strains of ferrets breeding in winter and permitting those properly conditioned, as to season, to continue to propagate. This would eventually preserve a race of ferrets breeding only in spring and summer, when light conditions bring sexual activity at a proper time for success in rearing young.

Both these females shed their winter fur in the two weeks immediately preceding the days on which they

were due to give birth to litters. The surviving one put on a summer coat which was apparently slightly denser and left smaller bare spots around the mammae than generally occur in oestrus or pregnancy of spring and summer. The new coat was about half grown or less when the young were born.

A third female, mated on January 11 with one of the same males, with abundant active sperm in the exudate from her vulva after mating, was only pseudopregnant and gave no young at term. Her winter fur began to loosen after February 7 and was shed rapidly from February 12 onward. She was moved to a warmer room and a warmer nestbox on February 10 so that the rigors of the other room would not have to be contended with; but shedding of winter coat was not the result of the warmer room, having been initiated before the change of room.

The behavior of the coats of these animals suggests that either low temperatures or a sudden shortening of the length of day by about six hours, at the time when the coat was growing out may have caused the incoming coat to be somewhat intermediate between that normal for summer and for winter. Since it is dangerous to generalize from only two cases, further tests under controlled conditions and with larger numbers are required. The fact is suggestive, however.

Summary

(1) Three ferrets were induced to come into oestrus in November, December and January by graduated increases in daily exposure to electric light after nightfall.

(2) They were mated once each with males rendered sexually active in the same way, and two became pregnant, the other only pseudopregnant.

(3) One of the pregnant females gave birth to a litter of six on January 14; the other died before parturition.

(4) The birth of this litter indicates that both male and female ferrets, brought into sexual activity in winter

by increasing exposures to light, are potent and fertile, and can produce young under conditions of temperature in which newly born ferrets were unable to survive.

(5) In each of these females winter coat was shed, beginning about two weeks before the expiration of term, and summer or spring nursing coat was completed after the termination of pregnancy or pseudopregnancy.

(6) It is suggested that numerous copulations within thirty hours may possibly cause luteinization of ovarian follicles without ovulation and prevent pregnancy in the ferret; whereas fewer or single copulations under such conditions more often lead to ovulations and pregnancies. This problem is being studied further with larger numbers and under controlled conditions.

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THE ECOLOGICAL DISTRIBUTION OF SOME NORTH AMERICAN ANURA[1]

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MANY of the recent distributional studies dealing with the North American Anura have, very naturally, been concerned with detailed consideration of limited areas (Pope and Dikinson, 1928; Ruthven, Thompson and Gaige, 1928; Smith, 1934; Schmidt and Necker, 1935, etc.). Others have shown the general distribution of individual forms (Wright and Wright, 1933; Stejneger and Barbour, 1923, etc.). While some of these authors have given notes of the habits and local habitats of individual species or subspecies, no one, so far as I can determine, has ever attempted to put the available data together to see if there is any broad correlation between the distribution of various types and the well-known climatic regions of the country.

The present paper is the result of an attempt to do this. As such, it does not deal with original observations upon the various Anura of the country but, rather, merely combines and evaluates data given in the literature and relates these data to well-known environmental regions. It is felt that perhaps something is to be gained and certainly nothing lost in taking a broad outlook of the United States as a whole from an ecological view-point.

The methods used were as follows: the general distribution as given in the literature of each American species or subspecies of the three main families (*Ranidae, Bufonidae* and *Hylidae*) was plotted on a map. The maps were then studied and the individual distributions compared with the general climate of the various sections of the country. The numbers of forms of each family in each of the climatic regions were then counted and tables

[1] Contributions of the Zoological Laboratory of the University of Oklahoma, No. 148.

constructed to show the result. Some difficulty was experienced in deciding just how many subspecies should be recognized, especially in the genus Pseudacris. It was soon found, however, that the exact number of forms recognized did not affect materially the general result.

It was found that the United States and adjacent areas in Mexico and Canada fell naturally into three regions, each tending to be dominated by one of the main anuran families. These are designated as (1) the frog area, (2) the toad area and (3) the tree-toad area and are shown on the accompanying maps (Figs. 1, 2, 3 and 4). The first

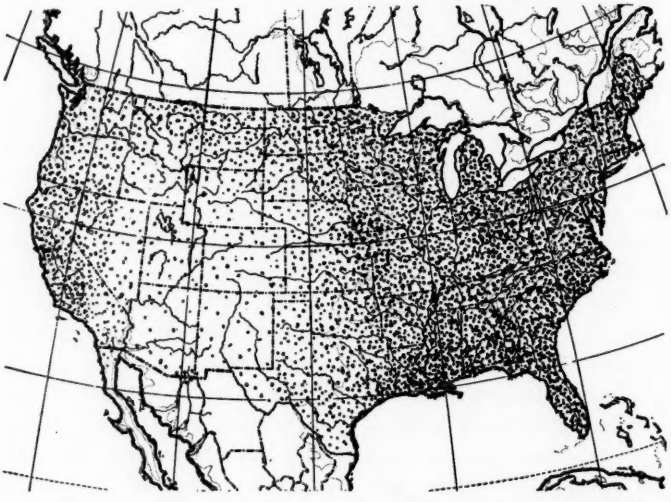

FIG. 1. Distribution of Ranidae within the United States. The darker the area the greater the number of species or subspecies. Plotted from data taken from the literature.

two of these are fairly clear-cut, but the last tends to overlap somewhat with the frog area to the north and east and, to a less extent, with the toad area on the west. It will be noted that the frog area comprises the East, Northeast, North and extreme West; that the toad area includes much of the Great Plains, especially toward the southwest; and that the tree-toad area is limited to the Gulf Coast and South Atlantic regions.

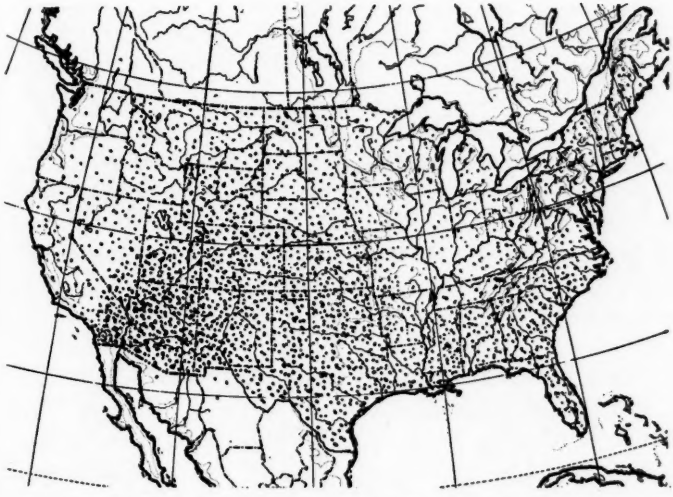

FIG. 2. Distribution of Bufonidae in the United States. Constructed in the same manner as Fig. 1.

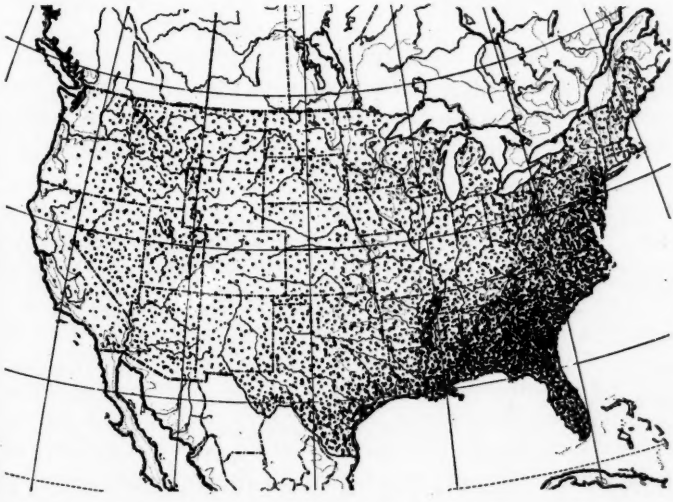

FIG. 3. Distribution of the Hylidae in the United States. Constructed in the same manner as Fig. 1.

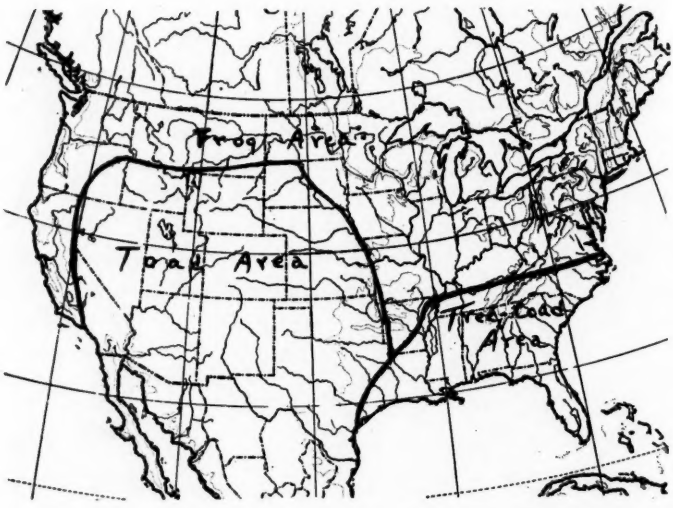

FIG. 4. The Anuran Areas of the United States. Constructed from inspection of Figs. 1, 2 and 3. The lines delimiting the various areas are necessarily but approximations.

Table 1 shows the number of species or subspecies of each family considered in each of these regions. The numbers in parentheses are the numbers of these forms which are wide ranging, *i.e.*, reaching well into other areas.

TABLE 1

	Ranidae	*Bufonidae*	*Hylidae*
Frog area	14 (3)	4 (2)	10 (4)
Toad area	7 (3)	11 (2)	9 (6)
Tree-toad area	9 (5)	4 (2)	16 (10)

If we eliminate such wide-ranging species as *Rana catesbeiana, R. pipiens pipiens, Bufo americanus americanus, B. fowleri, Acris gryllus, Hyla crucifer,* etc., all of which range through at least two of the indicated areas or reach well into two of them, we arrive at the number of forms of each family which are limited to, or nearly limited to, each of the three areas. Table 2 shows this.

TABLE 2

	Ranidae	*Bufonidae*	*Hylidae*
Frog area	11	2	6
Toad area	4	9	3
Tree-toad area	4	2	6

A glance at either table is sufficient to show that the areas outlined are real. It is well to note, however, that each area has peculiar to it at least one representative of each family. Examples of this are *Rana grylio, R. aesopus* and *Bufo terrestris* in the tree-toad area; *Rana areolata* and *Hyla arenicola* in the toad area; *Hyla andersonii, Pseudacris septrionalis, Bufo boreas boreas* and *Hyla regilla* (the last two overlapping somewhat with the toad area) in the frog area. In addition, wide-ranging species of all three families, of course, occur in all three areas.

If we consider individuals rather than species or subspecies, the tendency for one family to dominate a given region is still more marked, except in the tree-toad area. I can testify from my own experience that it is generally easier to find a frog than a toad or tree-toad in New England, upper New York or Wisconsin. On the other hand, in Oklahoma it is easier to find a toad, although *Acris gryllus* is locally very abundant at times. I have never collected in the tree-toad area, but judge from the literature that individual frogs and tree-toads are quite abundant, with toads less so.

If we inquire into the habits of frogs, toads and tree-toads, taking these from a general view-point, I believe that it can be shown that frogs are more dependent upon water than are toads. Tree-toads seem somewhat intermediate in this respect, but many of them have aboreal habits which tend to limit their distribution to those areas in which trees grow. It is of course realized that individual species differ somewhat in these matters; that the bullfrog, for example, rarely goes far from water; that *Rana pipiens* may travel the meadows long distances from water; that the two common wood frogs range the

woodlands of the North and East for weeks at a time without once entering water. Similarly, *Acris gryllus* does not climb trees and *Bufo alvarius* is semi-aquatic.

If one may be permitted to theorize a little, these and other similar exceptions tend to strengthen rather than to weaken the general conception advanced here. If frogs in general tend to be limited to places where water is available and if toads tend to replace them in drier situations, one might expect that in regions where few frogs can go toads would tend to become adapted in such a manner as to fill the ecological niches thus left vacant. This would at least explain the presence of a semi-aquatic toad, *B. alvarius,* in the drier portion of the Great Plains. A somewhat similar explanation might be advanced for the adaptation of the wood frogs for a land life beyond most others of their kind. The notable success of our most wide-ranging frog, *R. pipiens,* may be explained, at least in part, by its adoption of the habit of ranging the fields to feed. Another case in point is the greater abundance of the water-loving frogs of the bull-frog type (*R. grylio, R. clamitans* and *R. catesbeiana*) as compared to the spotted frogs in the swampy regions of the South. The general distribution of the tree-toads seems to indicate that they are much like the frogs in their dependence upon water; but they have, in general, another requirement—the presence of an abundance of trees. From this general point of view, we may look upon the *Hylidae* as frogs which have taken to the trees. Forms like *Acris gryllus,* so far as habits and habitats are concerned, are, from this point of view, tree-toads which have developed (or retained) a more frog-like habit than their fellows.

If we turn now to the general climates of the three areas outlined above, we find that the frog area lies almost wholly in that region of the country where most water is available. In the Northeast and East there is abundant rainfall throughout the year (compare Thornthwaite, 1931). In the north central area glacial lakes are abundant and evaporation rates are not excessively high. On

the Pacific coast, the proximity of the Pacific Ocean to high mountains causes rainfall and fogs. As compared with this, the toad area comprises the great Southwest, where rainfall is not only inadequate, especially during the summer months, but evaporation rates are high, due to higher temperatures. The tree-toad area lies in that region of the country having the most extensive swamp lands. Rainfall, humidity and temperatures are high and, therefore, trees are abundant. We should expect this region, therefore, to be dominated by tree-toads, but we should also expect many frogs, especially of those types which like water the most. This is probably the reason why the tree-toad area is not so clearly marked as are the other two areas and why frogs and tree-toads tend to be co-dominants in this region.

Incidently, it should perhaps be noted that the two other principal anuran families of the country (*Scaphiopodidae* and *Brevicipidae*) do not contain enough species and subspecies to show how they might fit into the above scheme. When individuals are considered, however, the spade-foot toads fall naturally into the toad area just as one would expect from their habits. Of the narrow-mouth toads, one species ranges from the tree-toad area well into the toad area and the other, *Gastrophryne texensis*, is limited to the toad area.

Since I have had no personal experience with the other two American anuran families (*Discoglossidae* and *Leptodactylidae*), I have made no attempt to relate these to the three areas outlined in this paper.

Summary

Detailed consideration of the general distribution of the forms of *Ranidae, Bufonidae* and *Hylidae* reported in the literature on North American Anura shows that the United States and adjacent regions may be divided into three areas, each tending to be dominated by one of these families, both as to individuals and as to species. Most frogs occur where most moisture is available, most toads

in the drier regions and most tree-toads where both moisture and trees occur abundantly. The tree-toad area is less distinct than the others because conditions there are also favorable for frogs.

It is clearly recognized that each region contains representatives of each of the above families, however, so that no area is exclusively a frog or toad area. Nevertheless, there is a tendency for the anuran groups to be segregated into the indicated climatic regions and for each to be adjusted, physiologically, to one of them.

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[2] Many other papers reporting distributions within limited areas were consulted. For a more complete bibliography, see the literature lists of the longer papers cited here.

A FREQUENTLY MUTATING GENE IN THE PINEAPPLE ANANAS COMOSUS (L.) MERR.[1]

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THE pineapple grown commercially in Hawaii has long been known as the Smooth Cayenne. The word "smooth" has reference to the leaves of the plant, which normally have a few small spines on the edges only at the tip and base. There has been a tendency in Hawaii during recent years to omit the word smooth when speaking of this variety, since there are no spiny varieties grown commercially.

Plants with spiny leaves, however, appear in Cayenne fields, and because of the inconvenience in agricultural operations they are undesirable. Consequently, a certain amount of rogueing is regularly practiced to discourage the increase of this type. Even with continual rogueing operations, which, it is admitted, are seldom thorough, spiny plants continue to appear. In addition to the spiny plants even a larger number are found having one or more leaves with spines and the remainder of the leaves normal.

All the wild pineapple species about which we have knowledge have spiny leaves. It is possible that the first smooth-leaved plant originated by a dominant gene mutation from the original spiny type. The spiny condition is recessive to the smooth of Cayenne, and plants of this variety are considered to be heterozygous for spines.

The spiny plants in a field of the Cayenne variety, on this hypothesis, represent a reversion to the ancestral type. It is difficult, if not impossible, to determine in Cayenne how many of the spiny plants and smooth-spiny chimeras are vegetative descendants of previously exist-

[1] Published with the approval of the director as Technical Paper No. 85 of the Pineapple Experiment Station, University of Hawaii.

ing ones which were not eliminated by the rogueing operations and how many may be new mutations in any given field population.

In this paper are presented some data from Cayenne and hybrid populations relative to the origin and genetic nature of the chimera and spiny plants which appear in populations of the vegetatively propagated, smooth-leaved Cayenne variety of the pineapple.

MATERIALS AND METHODS

In a casual examination of 19,441 plants growing on different plantations on Oahu, 0.26 per cent. were found to be spiny and 0.90 per cent. spiny-smooth chimeras. It is probable that some of the less conspicuous chimeras, such as spines on one half of one margin of a single leaf, were missed in this first examination. A later and more careful examination of 2,334 plant crop plants and 3,167 mature first ratoon shoots in fields at the Experiment Station revealed 1.02 per cent. spiny-smooth chimeras in the plant crop field and 1.45 per cent. in the first ratoon shoots. The "plant crop" results from the growth of slips (or other plant pieces) which are planted in the soil. In starting a plant crop, therefore, opportunity is afforded for the elimination of slips with spiny leaves. Ratoon shoots grow from the leaf axils of the plant crop plants and constitute a second generation of plants from the original planting. Rogueing is not practiced in ratoons.

The difference between these two later percentages may represent the chimeras removed by rogueing at the time the slips are gathered. When this difference, 0.43 per cent., is added to the percentage 0.90 found in the Cayenne plantations, the sum, 1.33 per cent., is still less than that found by careful examination of ratoon crop plants at the Experiment Station.

The chimeras may produce three kinds of progeny by vegetative propagation: plants with all leaves smooth, plants with all leaves spiny and chimeras of different proportions of spiny and smooth leaves.

The size of the spiny area of a chimera varies widely. There may be a spiny sector only a few inches long on a single leaf margin at a place where spines do not normally occur in the Cayenne variety, or as much as half the plant may have spiny leaves. In listing plants as chimeras, we have considered nothing having less than one half of one leaf margin with normally developed spines. Such mutations have occurred late in somatic development, while, on the other hand, mutations, resulting in a large sector of spiny leaves, occur relatively earlier in plant development. Only those mutations became evident, however, which occurred in meristem cells giving rise to leaf edges. Mutations may occur also in meristem cells which do not produce spines on the leaves. The mutation rate which we have obtained is therefore the rate in the portion of the meristem which gives rise to leaf margins.

Jones (1935) has called attention to the fact that recessive genes may appear in mosaics or chimeras in vegetative propagation due to chromosome aberrations of several kinds, but which, in general, serve to remove the dominant gene from a cell, thus permitting the single recessive gene to become effective in subsequent growth of the chromatin deficient cell.

Spiny Cayenne plants give no evidence of any chromosome deficiency in either their own development or in the progeny from crosses and it is therefore believed that their appearance is due to gene mutation, and not to chromosome irregularities.

The number of chimeras found in the plantations was approximately 3.5 times the number of spiny plants. Some of the chimeras may also arise *de novo* by a recessive gene mutation, and thus would not be related to a previous chimera.

It was not possible to determine from the plants on the plantations what proportion of the chimeras were due to recent gene mutations. Certain hybrid populations, however, appeared suitable for this purpose.

All the smooth-leaved plants in populations of hybrids produced by crossing smooth and spiny-leaved varieties

should be heterozygous for the gene for spines. Any chimeras in such populations could be ascribed to mutations in somatic cells subsequent to zygote formation, since all these hybrid plants grown from seeds began their growth as a single cell. The number of chimeras found in these heterozygous seedling plants, in the various Cayenne populations and in the homozygous clon 8341, are given in Table 1.

TABLE 1

THE NUMBER AND PERCENTAGE OF CHIMERAS IN POPULATION OF PLANTS HETEROZYGOUS AND HOMOZYGOUS FOR THE GENE FOR SMOOTH LEAF MARGINS

Culture	No. of plants	No. of chimeras	Percentage of chimeras	No. of leaves	Percentage of chimera leaves
Heterozygous hybrids ..	3,327	174	4.97	175,050	0.994
Plantation Cayenne .	19,441	174	0.887	980,750	0.018
Station Cayenne Field 5 ...	2,334	24	1.02	116,700	0.020
Station Cayenne Field 3 Ratoons ..	3,167	46	1.45	158,350	0.029
Homozygous Smooth, Clon No. 8341 .	867	0	0	43,350	0

The marked difference in the percentage of chimeras in the Cayenne ratoon plants, where no elimination of plants with spiny leaves has been made, and in the heterozygous hybrids was unexpected. By applying the fourfold table χ^2 test[2] given by Fisher (1932) for independence we get $\chi^2 = 66.18$, which is clearly opposed to the hypothesis that the two samples are from the same kind of population.

An explanation of the difference in per cent. of mutations in these two populations may be found in the hypothesis that a portion of the Cayenne population consists of S/S plants which do not produce chimeras. It has been shown that the homozygous smooth-leaved clon 8341 does

[2] $\chi^2 = \frac{(ad - bc)^2 \quad (a+b+c+d)}{(a+b)\,(c+d)\,(a+c)\,(b+d)}$, where a, b, c and d are the four observed numbers 3167, 46, 3327 and 174.

not produce spiny-smooth chimeras. A Cayenne plant with the genotype S/S could arise by gene mutation from the heterozygote S/s.

An alternative and less likely hypothesis to account for the observed difference in percentage of chimeras would be the presence of modifying genes in one population which would condition the mutation rate and their absence in the other. However, 25 of the 33 crosses from which the heterozygous hybrids were taken had Cayenne in the recent ancestry, and it is therefore not probable that the assumed modifying genes could be present in one population and not in the other.

The clon 8341, derived from a hybrid between two varieties, each heterozygous for the gene for spines, is homozygous for smooth leaves. When plants of this homozygous smooth clon are crossed with spiny plants all the progeny should be smooth in phenotype and heterozygous for the gene for spines. A few chimera plants and a few entirely spiny plants regularly appear in these populations of hybrids.

TABLE 2

EVIDENCE OF SOMATIC MUTATIONS OF S TO s IN S/S AND S/s PLANTS. THE S/s PLANTS BEING THE PROGENY OF S/S × s/s PARENTS

Cross	Progeny segregation					
	Smooth	Chimera		Spiny		
7789 (s/s) × 8341 (S/S) ♂	63	4	(5.97 per cent.)	4	(5.63 per cent.)	
8341 (S/S) × 7789 (s/s) ♂	201	6	(2.90 " ")	8	(3.72 " ")	
8341 (S/S) × Wild Brazil (s/s) ♂	68	1	(1.45 " ")	1	(1.43 " ")	
Totals	332	11	(3.20 " ")	13	(3.65 " ")	

The chimera, as we have already shown, results from somatic mutations which occur in the hybrid plant during growth. The spiny plants represent individuals in which the mutation of S/s to s/s took place in the one-cell stage of the zygote or were due to s gametes being produced by the S/S parent as a result of a somatic mutation in the parent plant from S/S to S/s which involved the meristem giving rise to gametes.

The percentage of chimera plants in Table 2 is determined on the basis of all those which started growth as heterozygotes, namely, the sum of the smooth and chimera plants. The percentage of spiny plants is determined on the basis of all the plants in the population.

The number of entirely spiny plants resulting from mutations of S/s to s/s in the population of 3,327 heterozygous hybrids listed in Table 1 could not be determined because these populations were segregating for homozygous spiny plants.

DISCUSSION

The high percentage of mutation of the gene S to the gene s places the S gene of the pineapple among that growing group known as frequently mutating or unstable genes.

Demerec (1932 and 1931) has studied the rate of mutation in somatic cells in *Drosophila virilis* and *Delphinium ajacis*. The miniature −3 gamma gene in *D. virilis* showed a percentage of mutation ranging from 4.51 to 9.92. This is comparable to the average of 4.97 per cent. found for the S gene in the pineapple.

The mutation rate in the *Delphinium* studies was perhaps even higher, but because the method of expressing the rate of mutation was on a different basis direct comparisons can not be made.

In all the cases of mutable genes studied by Demerec in the two genera mentioned above, the mutations have been from a recessive type to the dominant original or wild type. In the pineapple the mutations produce the wild-type character of spiny leaves, but the direction of the change is from a dominant to a recessive character.

An unstable gene producing chlorophyll spots on the leaves of 11.5 per cent. of the seedlings of the yellow-inconstant variety of the Japanese morning glory, *Pharbitis Nil*, is reported by Imai (1930). Miyazawa (1929) studied a similar strain of the same species which has such a high degree of instability that only about 25 per cent.

of the seedling progeny were like the mother plant. Although these plants were sexually propagated the mutations described occurred in somatic cells and were found to vary with different stages of ontogeny.

In the pineapple we have not been able to detect any differences in mutation rate with different stages of development. As has already been stated, a wide range of chimera types has been found, including as the extremes, spines on one half (usually the basal half of the leaf) of one margin of one leaf to approximately one half of the plant being spiny.

The appearance of entirely spiny plants in the progeny produced by crossing plants of the homozygous smooth clon 8341 with spiny plants is of considerable interest, for it indicates that mutation of S to s is taking place in the homozygote as well as in the heterozygote. There are two possible explanations for the appearance of these spiny plants among the progeny of a homozygous smooth parent. One is that the mutation from S/S to S/s occurs in somatic tissues producing linear areas of S/s tissue. When such areas are included in the meristem from which gametes are produced some s gametes result. The other possibility is that the mutation from S to s occurs in cells after the reduction division of the pollen and egg mother cells. In gametogenesis there are two divisions of haploid cells where mutations might occur before zygotes are formed. Reciprocal crosses listed in Table 2 show that gametes carrying the gene for spines are slightly more numerous in pollen than in the egg cells of the 8341 plants.

Demerec (1932) found in *Drosophila virilis* that the rate of mutation per gene was the same in heterozygous and homozygous females, and that in flowers of *Delphinium* the visible mutation rate was twice as high in plants homozygous as in plants heterozygous for the same gene. If such were the case in pineapples, then the mutation rate producing genes for spines from genes for smooth would be twice as great in homozygous S/S plants like 8341 as in the heterozygous S/s ones like Cayenne.

It is difficult to determine the rate of mutation in homozygous (S/S) cells of the pineapple, since the direction of the change is from the dominant to the recessive character, and the heterozygote is phenotypically like the homozygote.

The fact that the number of spiny plants in the progeny of homozygous smooth and spiny plant parents shown in Table 2 is approximately the same as the number of chimera plants may be considered an indication that the total mutation rate is also high in the homozygous cells, but we can not state whether it is higher than in heterozygous cells.

Even should the rate of total somatic mutation be higher in S/S than in S/s plants, chimeras are less likely to appear in the S/S plants, due to the necessity for a second gene mutation to occur in tissues in which one had already taken place. In 867 homozygous plants of the clon 8341, which were very carefully examined, not a single chimera was found. In the same number of heterozygous plants we should have found 43 chimeras.

The difference in the percentage of chimeras found in Cayenne plants, 1.45, and hybrid plants, 4.97, the latter all known to be heterozygous, is of considerable interest. The original smooth-leaved plant probably arose as a dominant mutation from the spiny kind. Among all the spiny pineapple plants of a number of different varieties which have been grown in our experimental plots for a number of years, not a single case of mutation from spiny to smooth has been recognized. It, therefore, appears that the change from S to s occurs much more readily than the reverse change of s to S. This is in line with the generally recognized fact that, in both plants and animals, dominant gene mutations are much less frequent than recessive ones.

The chromosome locus of the spiny gene in the pineapple is somewhat similar in its mutation behavior to the Bar eye locus in *Drosophila melanogaster*. A single Bar-eyed male fly was found by Tice in 1913 and according to

Sansome and Philp (1932) not a single mutation of normal to Bar has been seen in the millions of flies critically examined since that time. The reverse mutation of Bar to normal has occurred a sufficient number of times that its rate has been calculated. We have no evidence that the gene for the spiny character mutates to the gene for smooth other than the fact that smooth-leaved plants exist. Since the spiny character is common to wild pineapples we assume it to be the primitive type and that the smooth-leaved form arose as a dominant mutation.

The number of Cayenne plants which have been produced by asexual propagation since the variety was established as a clon with the genotype S/s (it can now be called a clon only by courtesy) is large enough to have permitted some mutations of s to S, even with an extremely small rate of mutations. Once even a single mutation of this kind had occurred the number of S/S plants could then be increased by vegetative reproduction.

It is possible in view of the difference in percentage of chimeras in known heterozygous plants and in the Cayenne variety that some of the Cayenne plants in the plantations are genotypically S/S and do not produce spiny-smooth chimeras in the same proportion as do the S/s plants.

The possibility of the presence of such homozygous Cayenne plants constituting a part of the Cayenne population is now being put to a genetic test by crossing a large number with plants of spiny varieties. The progeny of the spiny varieties when crossed with homozygous Cayenne plants should all be smooth in appearance. The progeny produced by crossing heterozygous Cayenne plants with spiny varieties should be equally divided between smooth and spiny plants. It should thus be possible, not only to determine whether homozygous smooth Cayenne plants exist, but also to isolate them and establish a clon in which spiny plants would be absent or the number very greatly reduced.

SUMMARY

The spiny-leaved plants and the spiny-smooth chimera plants which are found in Cayenne fields originate as somatic mutations.

The percentage of chimera plants was found to be smaller in the Cayenne variety, which genetic tests had indicated as being hybrid for spiny leaves, than in smooth-leaved hybrid plants produced by crossing smooth-leaved with spiny-leaved varieties. This difference, it is believed, may be due to homozygous smooth-leaved plants in the Cayenne population which do not produce chimeras.

A clon derived from a hybrid plant homozygous for smooth leaves contained no chimeras when a similar sized population of heterozygous plants would have contained 43 chimeras.

A method is outlined by which homozygous Cayenne plants can be identified if they exist in the variety.

The rate of mutation from smooth to spiny in the pineapple, 4.95 per cent., places this in the category of frequently mutating or unstable genes.

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STRUCTURAL AND CHEMICAL ASPECTS OF ANIMAL COLORATION[1]

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COLORS have always exerted an attraction for artists, scientists, laymen and savages alike the world over, and chromatology finds very real and wide use both in various pure sciences and in such applied fields as those of agriculture, certain industries and medicine. The experimental or practical geneticist who may be concerned with the inheritance of any of various color manifestations is really studying the heritability of certain definite kinds of metabolic processes; likewise the physician, who bases many of his diagnostic conclusions upon the colors of tissues, other structures and body fluids, is scrutinizing the signposts of various normal or aberrant metabolic functions. The colors manifested by animal organisms may be structural or pigmentary, and of the latter, either endogenous or exogenous, *i.e.*, derived either metabolically or from plant food, directly or indirectly.

We are reminded (Wood, 1934) that "opaque substances are seen by the light reflected from their surfaces; transparent substances in part by reflected light and in part by transmitted light," and that "the colors of most natural objects result from absorption." Incident light, penetrating to a greater or less degree the surface of an object, undergoes reflections, diffractions or refractions as it strikes minute internal structures; the absorbed rays which are totally reflected or refracted in the interior of a body, do not reappear but are converted into heat, while the residual light emerges minus these most strongly absorbed rays and we say that the object has a certain color. The principle of selective absorption of various constituent rays of white light holds, whether the re-

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sponsible structure of the material be microscopically visible, as in prisms, gratings or striae, or of the colloidal range of sizes, as in certain fluids, thin films or laminae (ultramicroscopically visible except in the region which merges with that of true crystalloids), or whether we are dealing with structures reduced in size to molecular dimensions, as in many colored inorganic and organic compounds. Needless to say, many color manifestations result from the combined effects of two or several such optical factors.

Structural Coloration

Total reflection of white light as such may be brought about by substances which, whether transparent or opaque, colorless or somewhat colored, are in a very fine state of division, the constituent particles being separated by a different medium such as air. Many substances may be nearly white, iridescent, yellow, red, blue, etc., or even black, depending upon such physical properties as the state of molecular aggregation, degree of subdivision, deposition in thin layers and orientation of the constituent particles. Examples of all classes are found in numerous colloidal systems, laminated deposits, amorphous or micro-crystalline powders and precipitated substances of the inanimate world.

We need not look far in the animal world for examples of the various grades of colored surfaces or combinations of them. However, we shall limit selected examples of purely so-called structural coloration to a few outstanding types, and shall then consider the true pigments, which are responsible for the great majority of animal colors.

Newbigin (1898), following Gadow's distinction between classes of colors, divides the structural colors of organisms into two main groups, *viz.*, those independent of, and those dependent upon the presence of a pigment. The first group may be classified as follows:

(1) Caused by total reflection (either white surfaces or natural prisms).

(2) Caused by surface striations (natural gratings).

(3) Caused by thin plates or laminae (natural films).

White aspects may arise from the separation of finely divided structural materials by air bubbles; examples of this are white hair or white feathers, the white fur of Arctic mammals, etc.; white may arise also from the deposition of certain compounds by some tissues, examples being colloidally dispersed, stored lipoid material, calcium carbonate in certain molluscan shells and in some crustacean carapaces, and guanin crystals in the iridocytes of many invertebrates and in the ventral skins of fishes. In certain instances, the same compounds may, by virtue of a definite crystalline habit, act as scintillating microprisms and bring about by refraction myriads of minute spectra or other iridescent effects; guanin in the skins of certain fishes contributes a silvery appearance or iridescent colors. Iridescence may also arise from the diffraction of light at finely striated surfaces, such as are observed in bristles, nails, feathers, spider-webs, cuticles of various arthropods or worms, bundles of striped muscle, etc. Instances of iridescence produced by thin plates or laminae are the calcareous mother-of-pearl lining of molluscan shells (wherein true diffraction also plays a part) and the mucous secretions of certain worms and mollusks. (See Poulton 1890; Newbigin, 1898.)

The brilliant blue and green structural colors are most frequently and conspicuously displayed in the feathers (and sometimes in the skin of body or face) of some birds, the wings, eyes and bodies of certain insects, the skins and eyes of many tropical fishes, and in the body surfaces of some marine gastropod and cephalopod mollusks. Such pure colors as well as the metallic colors observed in many birds and insects are due to the reflection of a part of the incident light by an optical surface, while an accompanying pigment layer absorbs part of the spectrum. It may be said that the greens result, in general, from a combination of a yellow pigment and a structural modification, while the blues, as well as the metallic and more somber color effects are associated with dark-colored pigments,

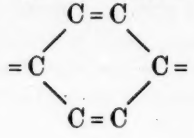
combined with optical surfaces. Such structures, of course, appear different by transmitted light, wherein the pigmentary effect alone is obtained. (See Newbigin.)

Structural or optical colors of organisms are recognizable by various tests. (See Newbigin, 1898, Verne, 1930.) They usually show variation either with the angle of vision of incident light, or when viewed by reflected versus transmitted light; extensive injury to the surfaces which exhibit the colors usually destroys the chromatic effect; immersion in a neutral medium of refractive index different from that of the natural medium also often causes the color manifestation to disappear; treatment of the colored structure with extractive reagents yields no pigment of a tint corresponding to the original color.

Color and Chemical Constitution

Regarding theories of color and chemical constitution, Porter (1924) writes, "There must be some fundamental property of matter that makes possible the development of color, and it must be a property that is dependent upon molecular structure and sensitive to slight changes in configuration—a property that makes possible a gradual and continuous shift from one end to the other of the visible spectrum through slight modifications of the molecule, either by addition or substitution, or through intramolecular rearrangement." Configurations in organic molecules which are associated with color are known as chromophores. Without representing the entire formulae of different dyes and other colored compounds, the following chromophores are given as examples (from Porter):

Double bonded carbon $>C=C<$ in carotenoids and in certain types of condensed rings such as indigoid dyes; double-bonded trivalent nitrogen $-N=N-$ between aromatic radicals, (in azo dyes); the quinoid ring



found in triphenylmethane dyes; nitro groups $-NO_2$, nitroso groups $-NO$, azoxy groups $-N(O)-N-$, and diketonic linkages $-C(=O)-C(=O)-$.

If, in addition to a chromophore grouping, a compound possesses certain polar groups such as amino ($-NH_2$) or hydroxyl ($-OH$) radicals in an aromatic nucleus, the colored compound is capable of becoming more or less permanently attached to textile fibers and is known as a dye. A radical which is thus responsible for the conversion of a colored compound into a dye is called an auxochrome. Thus, while azobenzene is a light yellow compound by virtue of its chromophore group ($-N=N-$) it will not dye fabric. Helianthin, or dimethylaminoazobenzene sulfonic acid, ordinarily known as methyl orange, however, contains the sulfonated auxochromic dimethylamino group, and is a dye. (See Porter.)

According to Lewis (1916) color is the result of selective absorption of certain light waves by valence electrons. Associated with the presence of chromophore groups in compounds is the modification of electronic vibration frequencies. Ordinarily possessed of frequencies higher than those of visible light, therefore absorbing only in the ultra-violet, the majority of substances show no specific absorption of visible light rays and so are without color.

If, however, in the words of Lewis, "either by a change in the constitution of the molecule or through a change in the environment, the constraints acting upon an electron become weaker," the vibrational frequency of that electron decreases, gradually approaching that of the limits of visible light. Whenever, therefore, this happens in the case of an organic substance, light waves of corresponding frequency (blue and violet) are absorbed (converted into heat) through frictional processes, and the emergent light is yellow. If the loosening of the con-

straint upon the electrons concerned is still greater, resulting in a further reduction in their vibration frequency, other colors will be manifested through a maximum absorption of light in the longer wave-length regions of the visible spectrum, and thus when absorption occurs in the orange and red, we have color gradations up into the complementary green and blue to violet regions. According to Bohr's view, light quanta of exactly the right magnitude (frequency) are absorbed when they bring about an electronic transition from an inner planetary orbit to an outer one. Should the reverse process take place, energy, instead of being absorbed, is now given up, and emission of light occurs. Earlier writers who adopted the view that absorption bands are due to the vibration of electrons were Erfle in 1907, Koenisberger and Kilchling, in 1909 and 1910, and Campbell in 1913. (See Watson, 1918.)

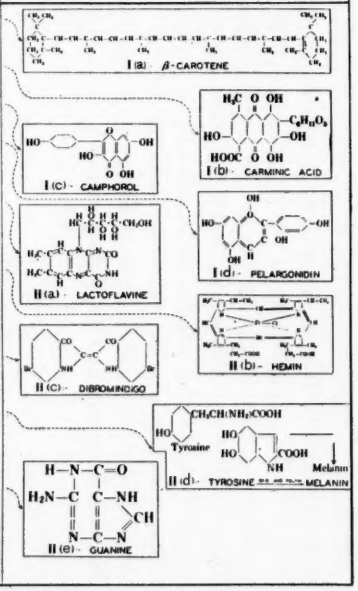
ANIMAL PIGMENTS

We might classify animal pigments in any of a number of arbitrary ways as Verne (1926) has done, *i.e.:* (1) with regard to chemical structure; (2) from a standpoint of morphology, including a consideration of tissues, cells or other structures wherein pigments are generated or stored, and the various physical states of pigments so distributed; (3) from physiological considerations of origin, metabolism and special rôles, or (4) according to color alone or to ascribed biological significance (*i.e.*, concealment, warning, etc.). We shall adopt the chemical classification as being the most fundamentally natural and shall attempt to consider briefly, in passing, certain of the more important physiological aspects of some of the pigmentary compounds. Ecological, morphological and hereditary aspects and certain biological significances of coloration in general, and of pigmentation in particular are treated by other contributors to this symposium.

ANIMAL PIGMENTS

CLASS	COLORS	ANIMALS	ANIMAL STRUCTURES	SOURCE	CHEMICAL ASPECTS	PHYSIOLOGICAL ASPECTS
I-NON-NITROGENOUS						
(a) CAROTENOIDS	RED, ORANGE, YELLOW	ALL PHYLA	SKIN, FEATHERS, EYES, CUTICLE, SERUM, EGGS, MILK, ETC	PLANT LEAVES, FRUIT, FLOWERS	FAT-SOLUBLE POLYENE HYDROCARBONS, ALCOHOLS OR KETONES	VITAMIN A GROWTH VISION, PERHAPS TRANSFORMATION OR STORAGE OF ENERGY
(b) ANTHRAQUINONES	RED, CRIMSON	SCALE INSECTS (COCCIDAE)	FAT BODY, EGG YOLK	PLANTS	WATER-EXTRACTABLE QUINOID DYES	
(c) FLAVONES	YELLOW	INSECTS (BUTTERFLIES)	WINGS	PLANTS, ESP. FLOWER PETALS,	WATER-EXTRACTABLE PHENYL γ-PYRONE DYES	
(d) ANTHOCYANINS	BLUE, VIOLET, RED	INSECTS	ALBUMINOID INCLUSIONS OF FAT CELLS	PLANT BLOSSOMS, FRUITS, AND LEAVES	WATER-EXTRACTABLE γ-PYRANE OR BENZOPYRANOL DYES	
II-NITROGENOUS						
(a) LYOCHROMES OR FLAVINES	YELLOW (GREEN FLOURESCENCE)	ALL PHYLA	VIRTUALLY ALL CELLS IN LOW CONCENTRATIONS	UNIVERSAL	WATER-SOLUBLE, HETEROCYCLIC CONDENSED RINGS CONTAINING -C=N- GROUPS	VITAMIN B ANTI-PELLAGRA ANTI-CATARACT? ANTI-ANEMIA? CELLULAR OXIDATION, WARBURG'S YELLOW RESPIRATORY ENZYME
(b) PYRROLIC COMPOUNDS	YELLOW, RED, GREEN, BLUE, BROWN	ALL PHYLA	ALL CELLS, BLOOD, SOME SECRETIONS (URINE, BILE, MANTLE GLANDS ETC.)	PLANT TISSUES	WATER-SOLUBLE BASES TETRAPYRROLIC (PORPHYRIN) COMPOUNDS USUALLY COMBINED WITH A METAL (Fe, Mn, Cu, Zn, V) AND OFTEN WITH PROTEINS AS WELL	RESPIRATORY (O₂ AND CO₂ TRANSPORT) PEROXIDE DECOMPOSITION (PEROXIDASES, CATALASES) CYTOCHROMES, EXCRETORY (BILE, URINE)
(c) INDOLE DERIVATIVES	BLUE, RED	MOLLUSCS, MAMMALS	MANTLE AND SHELLS OF SOME MOLLUSCS, OXIDIZED URINE OR EXCREMENT OF SOME MAMMALS	BREAKDOWN OF TRYPTOPHANE	WATER-SOLUBLE INDIGOID DYES	EXCRETORY
(d) MELANINS	YELLOW ORANGE, RED, BROWN, OR USUALLY BLACK	ALL PHYLA	EYES, HAIR, SKIN, INTERNAL MEMBRANES, MELANOTIC TUMORS, URINE, INK OF CEPHALOPODS, TISSUES OF CERTAIN COELENTERATES AND WORMS	DECOMPOSITION AND OXIDATION OF TYROSINE	INSOLUBLE OR WATER-SOLUBLE OXIDIZED POLYMERS	EXCRETORY, SECRETORY, LIGHT SCREENS? ENERGY STORAGE OR TRANSFORMATION?
(e) PURINES	WHITE, YELLOW	MANY INVERTEBRATES AND VERTEBRATES	INSECTS WING- AND BODY-MARKINGS, SPECIAL CELLS AND STRUCTURES OF VERTEBRATES	NUCLEO-PROTEINS	INSOLUBLE OR WATER-SOLUBLE PURINE COMPOUNDS (URIC ACID, GUANINE ETC.)	EXCRETION AND DEPOSITION PRONOUNCED DURING MATING SEASON
III-CHROMOLIPOIDS	RED, YELLOW, BROWN	MANY PHYLA	FAT DEPOTS	LIPOID SUBSTANCES	FAT-SOLUBLE OXIDATION-DERIVATIVES OF UNSTABLE LIPOID SUBSTANCES	RESERVE FOOD

REPRESENTATIVE COMPOUNDS



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The author wishes to designate the following writers, from whose works (*op. cit.*) the respective chemical formulae for the representative compounds named in the table were taken directly: β-carotene from Zechmeister (p. 36); carminic acid from Mayer (p. 88); camphorol from Mayer (p. 118); pelargonidin from Gortner (p. 620); lactoflavine from Harris (p. 340, there designated only as 6,7-Dimethyl-9-1-arabo-flavin); hemin from Mayer (p. 185); dibromindigo from Harrow and Sherwin (p. 509); tyrosine, melanin, etc., from Harrow and Sherwin (p. 506); guanine from Bodansky ("Introduction to Physiological Chemistry," Wiley and Sons, 1927, p. 293).

The principal pigments of animals, whether on the one hand originally pigments of plant origin, changed only slightly or not at all in the animal, or, on the other hand, peculiar to the animal kingdom, might be listed according to the chemical classification shown in Table I.

The pigments listed in all the first group (Ia, b, c, d) and in first member of the second group (IIa) may be regarded as of exogenous origin in general, while the rest are to be considered as mainly endogenous.

CAROTENOIDS

The red, orange and yellow pigments, known collectively as carotenoids, whose number of known compounds is constantly increasing, have such wide distribution throughout the living world as to be nearly universal. They occur in plants ranging from the most lowly organized, *i.e.*, bacteria and fungi, to the most highly developed spermatophytes, and in animals, from protozoa to the highest mammals, including man, although certain exceptions are encountered in the animal world, not only among the invertebrates but more particularly in some mammals. The carotenoids, or polyene pigments, may be classified chemically by the following properties. (See Zechmeister, 1934.)

(1) Light yellow to deep violet red in color.

(2) Two (or three) absorption bands in the visible spectrum mostly in blue to violet region.

(3) Soluble in lipoids and in the typical lipoid solvents; insoluble in water.

(4) More or less pronounced instability toward atmospheric oxygen. (Autoxidation accompanied by bleaching of color.)

(5) Resistance against treatment with alkalies.

(6) Dark blue or similar coloration with concentrated sulfuric acid; also usually but slight resistance against the action of acids.

(7) Carbon and hydrogen, or these elements plus oxygen, as the only constituents of the molecules; absence of nitrogen, as in the fats, waxes and sterols.

Structurally the carotenoids consist of long chains of unsaturated carbon atoms side-linked to hydrogen atoms or to methyl radicals, and arranged in conjugated double

bond systems, usually, although not always with an ionone ring at either end of the molecule. (Table: Ia).

The brilliant yellows, oranges and reds in many fishes, reptiles, birds and invertebrates are due to the presence of carotenoids. Many grays, browns and olive-greens in crustacean carapaces are the colors of carotenoids, such as the red acidic compound astacene, a specifically animal carotenoid chemically combined with protein. That carotenoids may be synthesized by animals from colorless compounds is accepted by some as a possibility, but the transformation has never been satisfactorily demonstrated. That they may be excreted unchanged, stored by animals changed or unchanged in various parts (skin, carapace, scales, serum, eyes, feathers, eggs, milk, etc.) or oxidized during metabolism to colorless compounds is well known. While it has usually been claimed that no transformation of carotenes into xanthophylls or *vice versa* is effected by animals, at least two workers in Europe (Virgin and Klussmann, 1932) believe, in spite of Palmer's strong evidence to the contrary (1922), that they have demonstrated a transformation of carotenes into xanthophylls by the hen. On the basis of some of our own experimental work (Sumner and Fox 1935; Fox 1936) we have concluded provisionally that the Pacific killifish *Fundulus parvipinnis* can oxidize hydrocarbon(s) of the carotene group to alcohol(s) of the xanthophyll series. We are all well acquainted with the fact that carotenes, or any of the carotenoids which possess the vitamin A precursor group that characterizes both halves of the β-carotene molecule, prevent nightblindness, xerophthalmia and increase resistance against infections of the respiratory tract. Xanthophylls are believed to play a possible rôle in growth processes (Rydom, 1933). Walls and Judd (1933) have summarized information concerning the occurrence of carotenoids in oil-droplets in the visual cones of the retina, where these pigments may serve as intraocular color filters. Vitamin A has been shown recently by Wald (1934, etc.) to play a bio-

chemical rôle in the retina during vision. According to Wald, visual purple (retinene), also known as rhodopsin, is a protein conjugated with Vitamin A. Xanthosis or carotenemia is a yellow condition of blood plasma and skin which follows the excessive ingestion of carotenoids; these compounds, however, are not known to have toxic properties. Future research will enlighten us further regarding their metabolism and special rôles. (See Lederer, 1935.)

ANTHRAQUINONES, FLAVONES AND ANTHOCYANINS

The anthraquinone derivatives, the flavones or anthoxanthones, and anthocyanins will be hardly more than mentioned in passing, since these compounds are characteristically plant pigments whose occurrence in animals is very limited, excepting, perhaps, in the case of the latter class. They are derived directly from plant materials used as food.

The anthraquinone derivatives are characteristically red or crimson water-extractable pigments found in certain homopterous insects among the Coccidae, notably lac, cochineal and wax insects of warmer climates. Found usually in the fat body or egg yolks, they are obtained in the form of potassium salts of acidic compounds, such as kermesic, carminic and laccainic acids. (See Verne, 1930.) (Table: Ib.)

The flavones or anthoxanthones, which are yellow plant pigments, phenyl derivatives of the γ-pyrone nucleus, also show rare occurrence in certain insects, notably the wings of certain butterflies which feed upon a plant source of the compounds. (Table: Ic).

The anthocyanins are the red, violet and blue compounds observed in the blossoms, fruit and leaves of many plants. They are chemically related to the flavones, being derivations of the γ-pyrane or benzopyranol nucleus and containing, unlike the flavones, an oxonium structure. (See Gortner, 1929.) They occur in certain dipterous and hemipterous insects and insect larvae, often in the

albuminoid inclusions of fat cells. Again these pigments are derived directly or but slightly changed from ingested plant material. (Verne, 1926.) (Table: Id.)

None of the above-named classes of pigments have been shown to fulfil a particular physiological function in animals.

The Lyochromes or Flavine Pigments

These pigments are distributed at relatively low concentrations in plant and animal tissues throughout nature. They are yellow, nitrogen-containing, water-soluble compounds which exhibit a green fluorescence. Heterocyclic rings, in which tertiary nitrogen-to-carbon linkages occur, supposedly impart the color to the molecule. The most interesting and important representative of this group is lactoflavin, which is now believed to be identical with Vitamin B_2, the anti-pellagra principle, which is believed to possess anti-cataract and anti-anemia properties as well. The flavin molecule has a function in cellular oxidation processes. It occurs both free and bound to protein or to purine bodies. The flavin-protein complex is identical with Warburg's yellow enzyme, which is said to be concerned with both oxygen transfer and anerobic respiration. (Harris, 1935.) (Table: IIa.)

Pyrrolic Compounds and Metallo-Proteins

There are represented throughout the animal phyla a great number of pigments of various yellow, red, green and blue colors whose molecules possess in common the pyrrolic nucleus, or more commonly the tetrapyrrolic grouping known as the porphin nucleus.

The most outstanding examples of this group are the hemoglobins, wherein iron is bound to the tetra-pyrrol ring (a porphyrin group) and the resulting compound, hemin, is conjugated with a protein of the globulin series. (Table: IIb).

Space does not permit much more than a categorical listing of some of the chief compounds of this great class, with occasional brief reference to physiological functions.

Far more detailed accounts of the pigments of this series may be found in the work of other reviewers and authors cited by them (Verne, 1926; Fischer and Newmann, 1932; Fischer and Orth, 1934; Mirsky and Anson, 1934; Harrow and Sherwin, 1935; Mayer, 1935). Hemoglobin, a red pigment, found in the bloods of all vertebrates and many invertebrates, acts as a respiratory aid in its function as an oxygen carrier.

Other pigments chemically similar to the hematins and hemoglobins are myochrome from muscle tissue, echinochrome, a brown pigment found in various echinoderms, chlorocruorin, a green one, also containing iron, encountered in numerous marine worms, helicorubin, a red compound found in liver and gut of mollusks and certain crustaceans, and actineohematin, a red dye which occurs in sea anemones. Pinnaglobin is a brown pigment whose molecule resembles that of hemoglobin, excepting that the former contains an atom of manganese instead of one of iron.

Other kindred respiratory proteins are: hemerythrin, an iron-containing pigment in the bloods of numerous marine worms; hemocyanin, a blue compound, chemically similar to hemerythrin, but containing copper instead of iron, found in many mollusks and crustaceans; hemosycotypin, a blue pigment (when oxidized) found in the gastropod Sycotypus, containing copper in the molecule, and vanadium chromogen, a brown organic compound rich in vanadium, found in the blood of ascidians.

Some metallo-protein pigments of animals are non-respiratory and of unknown functions. Examples are turacine, a copper-containing red-violet pigment in the feathers of certain birds, turacoverdine, a similarly occurring green pigment; and aplysiopurpurine, the purple dye believed to contain both iron and manganese, secreted by the mantle glands of the sea hare.

To the respiratory functions of hemin and derivatives of it, should be added mention of other physiological functions, such as the now established identity of catalase, the

hydrogen peroxide-decomposing catalyst, with a heme (an iron-porphyrin). The peroxidases are believed to be iron porphyrins also. Cytochrome, the universally occurring yellow oxidation catalyst encountered in all cells, was established as an iron-porphyrin derivative a decade ago.

Degradation products of hemin, which contain no metal but retain their pyrrolic character, are found in the bile of vertebrates or homologous fluids of invertebrates. Bilirubin and its oxidized form biliverdin, whose names suggest their respective colors, occur in the biliary fluids of vertebrates, while in various invertebrates, notably among the mollusks, are found analogous pigments such as helicorubin (containing iron) in the snail, derived from hematin compounds, and so-called hepato-chlorophyll in the hepatopancreas or gastric gland of certain other mollusks, a green pigment supposedly derived from the degradation of chlorophyll to give aetioporphyrin derivatives.

Bile pigments, such as biliverdin and bilirubin, appear in the urine only under certain pathological conditions. Urobilin, a brown-red pigment which appears in urine on standing, is closely related to the bile pigments and to similar compounds of common origin encountered in feces.

The pathological condition of true jaundice, with which carotenemia was at first erroneously confused, is marked by an extreme yellowing of skin, cornea and urine, accompanied by great lassitude, anorexia, constipation and paleness of feces, and is due to the accumulation of bile pigments in the tissues through obstruction of the ordinary excretory paths.

Indol Derivatives

The indol pigments are derived from compounds containing the phenopyrrol, or indol nucleus, notably the amino acid tryptophane, common to most proteins.

Indigo, the deep blue dye found in certain plants, as well as in some abalones, is a well-known derivative of

indole. Indole and skatole are largely responsible for the characteristic odor of fecal matter. Part of the catabolic indole is detoxicated in the body and excreted as indoxyl sulfuric acid, or indican, in the urine of man and of herbivorous mammals in particular. On oxidation, such compounds impart to voided urine reddish or blue colors.

Royal purple, also known as Tyrian purple, a purple of the ancients, is dibromindigo obtained from a gastropod *Murex brandaris*. Punacine and janthine are other examples of purple pigments derived from indole, and occurring in marine mollusks. (Table: IIc).

MELANINS

The melanins are a class of reddish, brown or black pigments that are very widely distributed in the animal kingdom. They are responsible for normal skin pigmentation and for the various colors of hair and of the iris. In addition, melanin pigments are encountered in the integument of vertebrates and invertebrates, in the lining of the coelomic cavity of many fishes, in melanotic tumors and in the ink secretions of cephalopods. Red or yellow so-called uranidines, found in sponges, corals, medusae and certain worms, are believed to be modified melanins. Urochrome, the principal yellow pigment of urine, is believed to be related to the melanins. In certain pathological or otherwise abnormal conditions, melanin precursors appear in the urine, causing it to darken on standing in air (melanuria).

The formation of melanin is related to the metabolism of the amino acid tyrosine, which occurs in protein material, or of closely kindred compounds such as dioxyphenylalanine (abbreviated to "dopa"). These phenolic amino acids react with oxygen in the presence of oxidizing enzymes, giving rise to oxidation products which polymerize to form either true melanins or melano-proteins, wherein a protein conjugant is associated with the melanin chromogen. (Table: IId).

Purine Derivatives

These compounds, which are usually white and sometimes yellow or other colors, have their origin in the degradation of nucleoproteins, and are characterized by the presence of a purine body in the molecule. Their sources are both endogenous (*i.e.,* derived from catabolic breakdown of cell nuclear materials, in particular) and exogenous (derived directly from ingested food).

The most commonly found animal purine is guanine, which occurs in the integument and serous membranes of numerous lower vertebrates (especially fishes) and invertebrates, often contained within special cells called leucophores, iridocytes or guanocytes. The choroid tapetum of certain mammals and birds contains guanine, which is said to play an important rôle in nocturnal vision. (Table: IIe).

The white and yellow markings of the bodies and wings of certain insects are attributable to the deposition of purine derivatives, notably leucopterin and xanthopterin, which are found in the hypodermal integument of wasps and hornets and in the wings of butterflies. According to Verne (1930), a remarkable correlation exists, notably in certain mollusks, batrachians, fishes and reptiles between internal purine metabolism and nuptial markings or coloration, at the time of mating. Verne suggests that, since such colorations are due principally to purine pigments, whose deposition is a result of the altered and increased nucleo-protein metabolism which characterizes spermatogenesis, there must be simultaneous elaboration and disintegration of endogenous nucleins, the presence of an excess of purine compounds leading to an increased deposition of the corresponding pigment in certain tissues.

There remains a large number of pigments whose chemical constitution is yet unknown. Noteworthy among these is the class called by Verne (1926) chromolipoids, which are often encountered in tissues or structures containing much lipoid material. For a discussion of these

pigments, Verne's work should be consulted. While this group of colored substances has in the past been confused with both melanins and carotenoids, they have been shown to be different chemically from either class. They are believed to be derived principally from unsaturated and often auto-oxidizable lipoid compounds, such as phospholipoids and fatty acids.

We have considered very briefly the chemical nature and some of the physiological aspects of a number of classes of colored compounds in the heterogeneous group of animal pigments. The existence of relatively loosely held electrons in such organic compounds is the cause of the manifestation of visible colors and is also the basis of chemical instability or reactivity. It is reciting a mere truism to state that the exhibition of a pigment by an organism is a display of some particular aspect of metabolism. But increasing biochemical information regarding the various compounds and structures exhibiting color will continue to extend our knowledge of the parts which they play in the metabolic economy of the organism.

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SHORTER ARTICLES AND DISCUSSION

HAECKEL, LAMARCK AND "INTELLECTUAL INERTIA"

In an informing article on "The Inheritance of Acquired Characters and the Provisional Hypothesis of Pangenesis," which appeared in the American Naturalist, Vol. LXIX, No. 724, 1935, we are told that a certain "statement of Haeckel's is little more than a justification for intellectual inertia." The writer quoted the statement in question from "The History of Creation," by Haeckel, Vol. I, page 78, edition of New York, 1896. It reads as follows: "' We have therefore no special occasion to examine the natural theories of development of the various Greek philosophers, since they were wanting in the knowledge gained by experience, both of organic and inorganic nature.' " Upon referring to a German edition of the above work by Haeckel, I found that the words quoted are an incomplete and also a somewhat imperfect translation of what Haeckel actually said. Moreover, when isolated from what precedes, they give an incorrect impression of his attitude.

According to the second German edition of the "Natürliche Schöpfungsgeschichte," Haeckel wrote as follows: "Wir haben daher auch hier keine nähere Veranlassung, auf die natürlichen Entwickelungstheorien der verschiedenen griechischen Weltweisen einzugehen, da denselben zu sehr die erfahrungsmässige Kenntniss sowohl von der organischen als von der anorganischen Natur abging, und sie sich demgemäss fast immer nur in luftigen Speculationen verirrten." These words may be translated as follows: "In this connection we therefore find no immediate reason for presenting the theories of natural development of various Greek philosophers, for their experiential knowledge of both organic and inorganic nature was very inadequate and they hence almost always lost themselves in airy speculations." In the English edition of this work the word *hier, i.e., here* or *in this connection,* and the concluding clause, which is both amplifying and explanatory, were omitted.

Haeckel had been considering *natural* in contradistinction to *supernatural* theories of development, and according to the English edition had said that the former are of much more recent origin and ". . . are met with only among nations of a more matured civilization, to whom, by philosophic culture, the neces-

sity of a knowledge of natural causes has become apparent; and even among these, only individual and specially gifted natures can be expected to have recognized the origin of the world of phenomena, as well as its course of development, as the necessary consequences of mechanical, naturally active causes. *In no nation have these preliminary conditions, for the origin of a natural theory of development, ever existed in so high a degree as among the Greeks of classic antiquity.*" (The italics are mine.) These words were followed by those already quoted from the NATURALIST and it is difficult to see how they make Haeckel guilty of "intellectual inertia."

All sufficiently familiar with the restless and stimulating and also provocative activities of Haeckel or with the criticisms and parodies such as "Die fünf Bücher Haeckel," directed against him during his own lifetime, realize that one guilty of intellectual inertia could not have provoked such things. Haeckel was an active investigator, teacher and protagonist, especially of evolution, Darwinism and monism. His words had the widest currency and he does not seem to have saved himself either physically or mentally in connection with what he had at heart. There were at least eight editions in German and four in English of the "Natürliche Schöpfungsgeschichte" alone. He was an advocate who often was very emphatic and sometimes dogmatic and uncritical or even credulous in regard to some things. His critics charged him with deliberate misrepresentation, but no one, so far as I am aware, has heretofore charged him with intellectual inertia in the sense of indolence. Outstanding students of his, such as Oscar Hertwig and Wilhelm Roux, vigorously defended him against the charge of deceit. Haeckel was guilty of airy speculations, but there surely is abundant evidence in his writings that he not only was appreciative of the older views in biology but also was very conversant with them and frequently referred to them. The many new terms introduced by him are not without significance in this connection.

In the article in the NATURALIST, it is further stated that "Its [Haeckel's statement] essential superficiality is shown by the fact that it contains a tacit assumption that Haeckel's own theories of development, and those of his contemporaries, were founded upon knowledge gained by experience. Haeckel was a convinced Lamarckian, etc. . . ." Since Haeckel had merely emphasized that the older philosophers had insufficient experimental knowledge for the particular subject under discussion, it

is hardly fair to imply that he held that experience played no part in their theories.

Although Haeckel was an avowed admirer of Lamarck and accepted many of his conclusions, it is incorrect to say without qualification that he was a "convinced Lamarckian." He accepted neither Lamarck's theory of adaptation nor his theory of inheritance in its entirety. On page 117 of "The History of Creation," Haeckel wrote:

> However, the way in which Lamarck wished to explain exclusively, or at any rate mainly, the change of forms, is after all in most cases not possible. He says, for example, that the long neck of the giraffe has arisen from its constantly stretching out its neck at high trees, and from the endeavour to pick the leaves off their branches; that as giraffes generally inhabit dry districts, where only the foliage of trees afford them nourishment, they were forced to this action. In like manner the long tongues of woodpeckers, humming-birds, and ant-eaters are said by him to have arisen from the habit of fetching their food out of narrow, small, and deep crevices or channels. The webs between the toes of the webbed feet in frogs and other aquatic animals, he says, have arisen solely from the constant endeavour to swim, from striking their feet against the water, and from the very movements of swimming. Inheritance, he says, fixed these habits on the descendants, and finally, by further elaboration, the organs were entirely transformed. However correct, as a whole, this fundamental thought may be, yet Lamarck lays the stress too exclusively on *habit* (use and non-use of organs), certainly one of the most important, but not the only cause of the change or forms. Still this can not prevent our acknowledging that Lamarck quite correctly appreciated the mutual cooperation of the two organic formative tendencies of Adaptation and Inheritance. What he failed to grasp is the exceedingly important principle of "Natural Selection in the Struggle for Existence," with which Darwin, fifty years later, made us acquainted.

Regarding the other tenet of Lamarckianism, the one regarded as most representative of him at present, the inheritance of acquired characters, Haeckel wrote on page 222 of the same work: "Which of the changes acquired by an organism are transmitted to its descendants, and which are not, can not be determined *a priori*, and we are unfortunately not acquainted with the definite conditions under which the transmission takes place." I feel prompted to add, however, that he immediately negated the effect of these judicial words and showed his credulity by adding, page 223, that "A few years ago a case occurred on an estate near Jena, in which by a careless slamming of a stable door the tail of a bull was wrenched off, and the calves begotten by this bull were all born without a tail." (!) No one will deny that such an accident may have occurred or that calves may have been

born tailless, but it is strange that such a man as Haeckel should relate the two as cause and effect. However, it is only fair to say that these words are not truly indicative of Haeckel's attitude toward heredity. Nothing could better reveal that than the concluding paragraph in the chapter from which the above quotations were taken. In the fourth edition in English, which was a translation of the eighth German edition, the anonymous translation of which was revised by E. Ray Lankester, the last paragraph reads as follows:

> Our knowledge of inheritance and propagation has made extraordinary progress during the last thirty years through the above and numerous other investigations. It is true none of the five above theories [his own] of molecules altogether explain[s] the enigma of these wonderful processes; their merit lies rather in having brought us to a clear consciousness of our inability to comprehend the immensely complex nature of these invisible processes. However, they have enabled us to cast aside the former mystical ideas as to their nature, and we have arrived generally at the conviction that we have here to deal with physiological functions, with the vital activity of cell-life, which, like all other phenomena of life, have to be traced back to chemico-physical processes; in fact, have to be explained by a mechanical method.

Since some historical aspects of the so-called law of recapitulation were considered in an article in the December number of the *Quarterly Review of Biology*, I will not comment here upon the statement that Haeckel was ". . . the inventor [*sic*] of the biogenetic law."

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HORMONES AND EVOLUTION

THE Biogenetic Law, though greatly modified in later years from its first enunciation, still gives a thumb-nail picture of the development of the individual, with the simple germ cells representing the remote protozoan ancestors, the complex mammal, its highest stage of development. In the study of the underlying causes regulating the complexities of the mammalian machinery, hormones have come to occupy an outstanding position. The presence of such hormones among the invertebrates is a moot question. Certain facts have been brought out in the study of the hormones of the anterior pituitary and their relation to the gonads of the rat, which suggest that hormones of this type probably do not exist in the lowest invertebrates, and also that they are unnecessary. Furthermore, these studies show that, in the long process of evolution, the germ cells of the mammal have not lost the ability to carry on certain functions without the aid of the hormones of the anterior pituitary.

These studies consist of an exhaustive morphological survey of the entire ovaries of 76 rats, cut in serial sections, in which the ova, primordial follicles, larger follicles and corpora were counted (Swezy, 1933). The rats which furnished these ovaries were of various kinds: normal, pregnant, pseudopregnant, hypophysectomized, thyroidectomized, and after administration of pituitary hormones in various ways for varying periods. The ova and primordial follicles, that is the ova with a single row of follicle cells, are included under one caption, as they reacted alike to the different stimuli. The number of ova and primordial follicles in some of these ovaries is given in Table I.

As shown in Table I, the entire absence of pituitary hormones, through the removal of the pituitary, causes an increase of about two times in the number of ova produced. In the earlier paper evidences were given to show that this is an actual increase and not a slow accumulation over long periods of time. So far as is known at present, the germinal epithelium is the only part of the body to continue its function of producing new cells at an increased rate after hypophysectomy. Nor is there any evidence that mitotic cell division actively occurs in any other tissue than the germinal tissue in these subnormal animals. The follicle cells are sister cells of the ovum (Evans and Swezy, 1931), and in these animals continue to divide mitotically, at a rate about equal to that in the normal animal, until the small follicle

TABLE I

NUMBER OF OVA IN THE OVARY OF THE RAT

From Swezy, 1933

Rat	Age	Type of rat	Ova and primordial follicles
1272	208	Normal	1995
1353	206		2030
1290	207		1858
1285	297		1615
8643	188	Hypophysectomized 64 days	4969
1141	189	62 days	4715
1081	202	79 days	3605
1130	202	90 days	4345
1196	153	2 rat hyp. daily— 9 days	1340
1996	182	8 rat hyp. daily—10 days	1570
1999	182		1447
2974	181		1472
2696	153	½ cast. ♂ r. hyp.—20 days	1450
6977	255	½—1 cc ext. daily—75 days	5490
8040	255	80 days	4826

may have six or eight rows of cells. After this degeneration is rapid. Further growth is undoubtedly dependent upon the presence of the pituitary hormones.

It was postulated in the earlier paper (Swezy, 1933) that the pituitary hormones had a depressing effect on the production of new germ cells. This is shown in the slightly lower count in the rats which had been given implants of rat hypophyses daily for periods ranging from 9 to 20 days. Even when hypophyses of castrated male rats were used this was true. Other conditions in the ovary were shown to support this conclusion. The last two rats in Table I show an increase equal to that of the hypophysectomized group. These had been given an acid extract of anterior pituitary, the early form of the growth hormone, which seemed to have a depressing effect on the rat's own hypophysis, as the ovaries were like those of the hypophysectomized group for the most part.

It is well known that follicles of the typical mammalian type, with many layers of cells, are not found among the lower animals, and among the invertebrates the number of follicles or

nurse cells may range from one or none in the lower forms to a complete row of cells surrounding the egg in some of the insects. Starting from the ovum, the first true mammalian structure to develop is the follicle of many layers of cells. In the hypophysectomized rat, the ovum or egg is able to continue its development without pituitary hormones until the complete formation of this first true mammalian structure is due to appear, when, lacking these hormones, the egg degenerates. It would seem that the forces or hormones necessary for this early development are not furnished by its environment but by the internal economy of the egg itself, as must be the case with the protozoa and lower invertebrates. It is also evident that the long period of evolution from amoeba to man has not resulted in the loss or change of this fundamental characteristic of the egg in its early development, surrounded though it has been by many and complex factors which tend to make it entirely dependent on its environment.

Within a few days after the operation, the ovary of the hypophysectomized rat becomes filled with atretic and regressing tissue, composed of follicles and corpora, and shrinks to the size of the infantile ovary. It is quite likely that this regressing tissue in itself affords an unhealthy milieu for the development of new germ cells, added to which are the metabolic disturbances attendant upon the loss of the pituitary, which are severe in all parts of the body. That the germinal epithelium is able to initiate the growth of new germ cells, which are then able to continue their own development under these adverse conditions, indicates a remarkable independence of its environment on the part of this germinal tissue. Under more favorable conditions of food supply, the results might be startling. The idea that the gonads are parasites of the body may not be far from the truth.

OLIVE SWEZY

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THE MACRONUCLEUS OF THE STENTORS

Out of fifteen texts and reference manuals consulted, including some of those most likely to be available to the beginner, ten give the impression through statement or figure that the macronucleus of *Stentor* as a genus is beaded. According to Stokes (1896), all the species of this protozoon are common. Data tending to confirm the statement have been obtained locally.

Johnson (1893) gave as the three principal forms for the nucleus in ciliates, meaning the vegetative or macronucleus it would seem, spherical or oval, vermiform and moniliform or beaded. A survey of the protozoan literature shows that the form of the macronucleus in *Stentor* is variously described as rounded, band-like, elongate, ellipsoidal, thread-like and beaded. The latter condition is most frequently mentioned and pictured.

The vital dye safranin was used as a stain for the study of specimens of *Stentor* in an exceedingly populous wild culture obtained during the spring months when drainage water was abundant and pools numerous. Some minutes after the application of the dye to the first example, a spherical macronucleus began to appear clearly outlined, approximately a third of the way down the body. It eventually colored a deep pink. Its diameter in the two right-angled axes was 23.33 μ. In a second instance the measurement was exactly the same. This shows that the macronucleus under these conditions was, in two cases, unquestionably very nearly, if not quite, spherical. In two additional specimens the larger measurements of 26.64 μ and 29.97 μ were secured. The nucleus was still globular, however.

With properly adjusted light the spherical macronuclear area was clearly visible under the dissection binocular and the lower power of the compound microscope. The characteristic position of the nuclear body made it unlikely that it was mistaken for the contractile vacuole. No case of the chain type of nucleus, so-called, was seen in this culture out of the large number inspected. It is true that previous to division the nodes of the moniliform nucleus coalesce. They produce a roughly spherical mass, however, and remain in that condition a relatively short period of time. The smooth, regular outline of the macronucleus and its very evident sphericity in isolated, free-swimming individuals as well as in those not completely separated would seem to preclude the possibility that the rounded form was only a temporary phase. The globular form has been reported by other investiga-

tors sufficiently often to establish its probable legitimacy as a constant feature of certain species.

Using the populous culture of this report as a basis, one would reasonably conclude that the Stentors of the moniliform nuclear category had received undue publicity and that it is incorrect and confusing to cite one shape only for the genus as being characteristic, to the exclusion of another or others which may be equally "typical."

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GRIZZLE, A COLOR CHARACTER OF THE BLACK RAT

THE grizzle or silver color variety has been reported in numerous mammals, both wild and domestic. Among the common rodents it has been described by Hagedoorn (1911) and Dunn and Thigpen (1930) in the house mouse, *Mus musculus;* and in the deer mouse, *Peromyscus maniculatus,* by Sumner (1932). Castle (1925) described a silver character in the domestic rabbit. The well-known gray color of horses seems to have much in common with these characters also. The purpose of this paper is to put on record observations which have been made on a grizzled color type in a stock of the black rat, *Rattus rattus rattus,* and the roof rat, *Rattus rattus alexandrinus,* maintained in the laboratory. There are presented notes on (1) description of the character, (2) conditions encouraging its appearance in the individual, (3) frequency in the population and (4) mode of inheritance.

The colony of rats which supplied the material was maintained in part by the Bussey Institution of Harvard University and in part by the University of Michigan. Grants from the Faculty Research Fund supported the work at the latter institution.

Grizzle or silver in the black rat and its relative, the roof rat, is very similar in appearance to the corresponding characters of the house mouse, deer mouse and rabbit. It appears in a series of types ranging from a very slight departure from the normal, full pigmentation to an almost silver pelage. The characteristic appearance is due to varying numbers of partially or completely

white hairs interspersed with colored ones in the pelage. The distribution of the white is uniform over the coat in complete pelages. During molting, however, two pelages of unequal degrees of grizzle produce irregular patterns. The pigmentation of the skin and other tissues does not appear to be affected. Each of the various color types of the black rat described by Feldman (1926) is subject to the effects of the grizzle. It has been observed in agouti, dominant black, recessive black and dilute individuals. It is very difficult to identify in the cream-colored type.

The grizzle character in the black rat, as in mice, rabbits and horses, was correlated with age. It did not appear in any individual before the age of six months and in many only after the age of one year or more. The rats were not classified as non-grizzled until they had passed the age of eighteen months without having shown white in any pelage. The average length of life in the colony was slightly more than two years. Most of the rats showed more pronounced grizzle in each successive pelage. Certain individuals, however, exhibited the reverse and became darker in one or more pelage. This fact indicates that aging alone does not produce the loss of pigment; but that other physiological conditions, probably induced by the endocrines, are the actual causes. The degree of pigment deficiency in the black rat and in other grizzled types is apparently one manifestation of a disturbed physiology which usually accompanies advanced age. The existence of animals at decreased temperatures has been advanced as the cause of silvering. These rats were kept in rooms which were maintained at about 70 degrees Fahrenheit. Temperature does not appear, therefore, to be a variable affecting the pigmentation in them. Selection may alter the age of inception and it may also produce uniformity with respect to age of inception and extent of whitening. In this stock of rats the degree of the grizzle was correlated with reduced general vigor and reproductive capacity.

The character appeared in the colony originally in 21 individuals among a total of 1,144 rats, of which 722 were of pigmentation sufficiently dark to allow the grizzle to be readily recognized. Of the non-cream individuals, therefore, 2.9 per cent. were grizzled. The average age at which the grizzle first appeared in the original 21 cases was 16.8 months. The population in which the first cases were observed was descended from ten rats from England, three male black rats from Wilton, New Hampshire, and two female roof rats from Baton Rouge, Louisiana. Later,

a pair of wild black rats from Wilton, New Hampshire, provided a new and plentiful source. The male of this pair and eight of the fifteen progeny from it proved to be grizzled. The average age of inception in this group was 12.7 months. The descendants of these black rats and four grizzled animals from the main colony were used for subsequent study of the character.

Selective breeding was first used to lower the age of inception to an average of 8.1 months and simultaneously to increase the degree of silvering. The fourth generation thus derived consisted of 57 rats, all of which were grizzled at the age of 12 months to the extent of about 50 per cent. of white hairs. Some of these were mated with non-grizzled rats from the main colony. None of the 108 progeny showed any grizzle at the age of 18 months. From them a second generation consisting of 223 rats was reared to the age of 18 months. Of these 50 became grizzled before that age. The age of inception varied between 7.1 and 17.8 months, with an average of 14.2 months. The degree of silvering among the second generation varied from roughly 5 to 75 per cent. of white hairs. These results seem to show that the grizzle character of the black rat is a recessive mendelian character which is quite variable in its extent and age of inception. It corresponds closely to the silver house mouse, grizzled deer mouse and silver rabbit in its mode of inheritance.

The species, *Rattus rattus,* has demonstrated a considerable capacity for producing variations. The following color types have been recorded: Black extension series of three allels—dominant black, agouti and cream or yellow; agouti series of three allels—white-bellied agouti, gray-bellied agouti and non-agouti; cinnamon or brown; albinism; "blue" dilution; red-eyed yellow; piebald; and grizzle. In addition a "waltzing" type has been reported.

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